

IDIOPATHIC SCOLIOSIS: AETIOLOGY, NATURAL HISTORY AND TREATMENT.

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All the work included in this thesis constitutes my own ideas, concepts and hypotheses concerning spinal deformities. In addition, the majority of these investigations, particularly the early ones have been performed by me alone. The development of the low dose radiographic technique for the assessment of scoliosis in children was necessarily performed in conjunction with expert radiation physicists. Some of the more recent work in Leeds has been performed in conjunction with several outstanding young orthopaedic surgeons in training with whom it has been a privilege and pleasure to collaborate. Their names appear on the relevant publications.

SCHOOL SCREENING FOR SCOLIOSIS : COHORT STUDY OF CLINICAL COURSE.

Dickson RA, Stamper P, Sharp AM, Harker P.

British Medical Journal; 281: 265-267, 1980.

This was the first longitudinal cohort study of spinal deformities in the community in the United Kingdom. Importantly, inconsequential spinal deformities secondary to a leg length inequality were identified as a potent cause of body asymmetry and were differentiated from true idiopathic scoliosis. Prevalence rates and progression potential of the different types of spinal deformity were established. All previous epidemiological surveys had not differentiated between true structural scoliosis and that caused by leg length inequality and their inferences were thus inexact.

School screening for scoliosis: cohort study of clinical course

ROBERT A DICKSON, PETER STAMPER,
ANNE-MARIE SHARP, PAUL HARKER

Summary and conclusions

A visual examination of 1764 Oxford schoolchildren for scoliosis was followed by low-dose radiography of the spine in those who showed evidence of asymmetric body topography; radiography was repeated a year later to assess progression. Forty-four children had curves of 10° or more. Two had a congenital abnormality and the remaining 42 were classified according to the type of curve: sacral tilt (compensatory), spinal (idiopathic), or combined (sacral tilt and spinal). Progression occurred in 6 (14%) children, none of whom had only a sacral tilt.

These results suggest that only by measuring sacral tilt can benign compensatory curves be differentiated from true idiopathic scoliosis.

Introduction

Without careful scrutiny of the community most children with progressive scoliosis remain undetected, only to present in later life with permanent disability.¹ Progression can, however, be prevented by early detection and effective conservative treatment while the deformities are mild and flexible.² Only 10% of curves detected by screening are progressive, most being non-progressive lumbar curves. The characteristics of deformities

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that progress and those that are benign are not clear, but compensatory non-progressive scolioses are commonly produced by a tilt of the sacrum secondary to inequality in the length of the legs.³ This cohort study of the clinical course of scoliosis is based on the observation that in many cases of benign scoliosis detected by school screening the curve is compensatory to a tilt of the sacrum but that this tilt is a result of an inherent developmental problem of the pelvis itself rather than any inequality in the length of the legs.

Patients and methods

A senior physiotherapist experienced in spinal disorders screened 1764 13 and 14 year olds in five Oxford schools for the presence of a spinal deformity by visual inspection with the patient standing erect and leaning forward (the "one-minute school screening test").⁴ Those showing evidence of asymmetric body topography were examined with low-dose spinal radiography⁵ in the erect position. The radiographs were measured by Cobb's method,^{6,7} and those children with scolioses of 10° or more were examined clinically to exclude a musculoskeletal disease as a cause of the deformity. One year later these children were re-examined and radiographs taken using the low-dose technique in the presence of two of us (RAD and PS) to eliminate postural variation.

Examination of the initial standing radiograph showed that many children seemed to have a sacral tilt with reference to the horizontal fluid level in the stomach included in the same radiograph (fig 1). The low-dose spinal radiograph taken at follow-up therefore included a contrast-medium spirit-level⁸ placed on the x-ray cassette just below the femoral heads. The site and magnitude of any scoliosis and the sacral tilt were recorded from these films. These data were compared with those of the initial low-dose radiograph to detect progression of the curve (a curve reaching 20° or more). Significances of differences in the mean size of the curves were confirmed statistically using Student's *t* test. The relation between magnitude of sacral tilt and magnitude of scoliosis was confirmed by deriving the correlation coefficient, *r*. Observations concerning proportions of type of scoliosis in different sites in the spine and observations concerning progression were confirmed using the χ^2 test.

Results

Of the 1764 children screened 147 (8.3%) showed evidence of asymmetry of body topography and 121 of these (6.9%) had radiographic evidence of a scoliosis. Seventy-seven children (4.3%) had curves of less than 10° but 44 (2.5%) had scolioses measuring 10° or more. The mean age of these 44 children (32 girls, 12 boys) was 14 years, 2 months (range 13 years, 4 months to 15 years, 2 months). One child had a double structural curve, and therefore the 44 children had 45 curves among them.

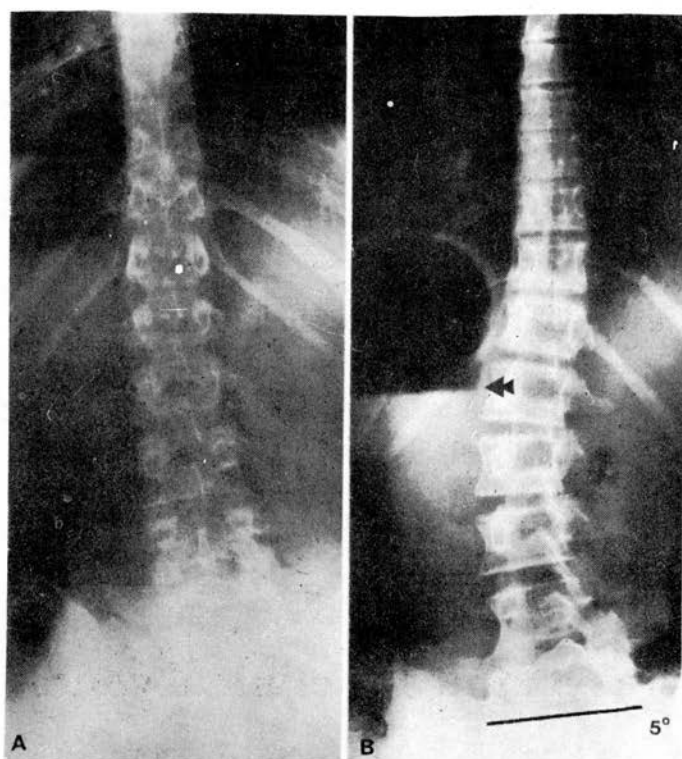


FIG 1—(a) Posteroanterior radiograph of thoracolumbar spine; x-ray cassette was inappropriately placed in holder, and there was no discernible horizontal reference level to compare with upper border of sacrum. (b) Posteroanterior radiograph of thoracolumbar spine; horizontal gastric fluid level was sharply demarcated (arrowed), and upper border of the sacrum was tilted 5° ; there was a compensatory lumbar scoliosis above.

TABLE 1—Numbers (percentages) of children with scoliosis

Type of scoliosis		No (%)
Non-genital		
Sacral	Sacral tilt only	9 (21)
	Sacral tilt and hip tilt	6 (14)
Spinal	Idiopathic only	18 (41)
Combined	Idiopathic and sacral tilt	4 (9)
	Idiopathic, sacral tilt, and hip tilt	2 (5)
	Idiopathic and hip tilt	3 (7)
Congenital		2 (5)
Total		44 (100)

SITE AND AETIOLOGY OF THE SCOLIOSES

Eight curves were thoracic, 11 were thoracolumbar, and 26 were lumbar. In two children the scoliosis was a result of congenital bony abnormality of the spine (unilateral failure of segmentation) but in the remaining 42 children no vertebral abnormality could be detected

radiographically. Twenty-six children (62% of the non-congenital cases) had a tilt of the sacrum in the direction of the convexity of the curve. In the remaining 18 cases the upper border of the sacrum was horizontal and the scoliosis was inherent to the spine itself. Three types of scoliosis could therefore be distinguished: sacral tilt, spinal, and combined (sacral tilt and spinal) (table I). In nine of the children with a sacral tilt there was no discrepancy in the length of the legs and the obliquity of the upper border of the sacrum was entirely due to pelvic asymmetry (fig 2). In six the obliquity of the sacrum was a result of a combination of pelvic asymmetry and a leg-length discrepancy. The sacral tilt in the children in the combined group was either because of pelvic asymmetry, leg-length discrepancy, or a combination of the two.

TYPE OF SCOLIOSIS AND SITE, CURVE MAGNITUDE, AND SACRAL-TILT MAGNITUDE

All scolioses in children in the sacral-tilt group were situated in the lumbar region and the lower end-vertebra of the curve was the sacrum itself. In those in the spinal group only two curves were in the lumbar region, the remainder being either thoracic (seven children) or thoracolumbar (nine).

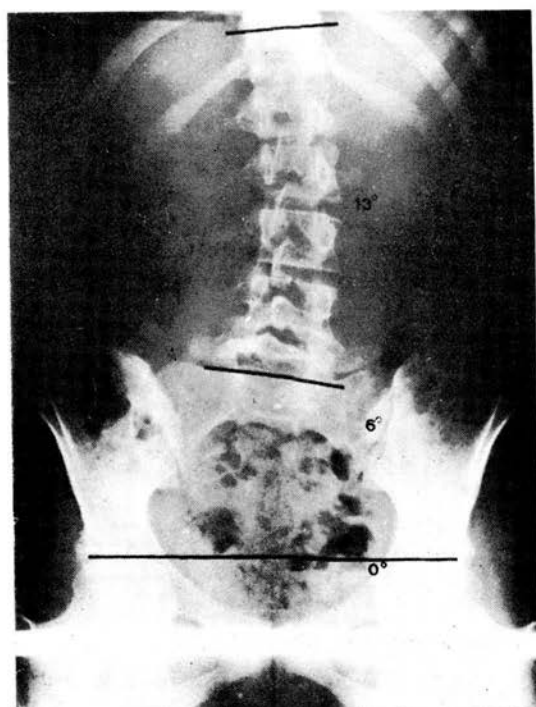


FIG 2—Posteroanterior radiograph of lumbar spine, pelvis, and hips incorporating contrast-medium spirit level just below femoral heads. There was no leg-length discrepancy but sacrum was tilted 6° and there was compensatory lumbar scoliosis above.

CURVE PROGRESSION

Progression of the curve was noted in 14% of the children with non-congenital scolioses. Progression did not, however, occur in those in the sacral-tilt group and deterioration was noted only in children in the spinal and combined groups (table II). Progression was noted in 38% of all thoracic curves, 18% of thoracolumbar curves (fig 3), and only 4% of lumbar curves; these differences in proportions were significant ($p < 0.05$).

TABLE II—Curve magnitude (as means \pm SD) in relation to sacral tilt (given as means \pm SD) and progression in three groups of children with different types of scoliosis

Type of scoliosis	Curve magnitude (degrees)	Sacral tilt (degrees)	Curve: tilt ratio	Curve: tilt correlation	No (%) with progression
Sacral	11.9 \pm 1.9	5.9 \pm 0.9	2:1	$r = 0.8$; $p < 0.001$	
Spinal	14.2 \pm 4.7	0			4 (22)
Combined	14.9 \pm 2.4	3.7 \pm 1.3	4:1	$r = 0.45$; NS	2 (20)

NS = Not significant.

Discussion

The prevalence of scoliosis in Oxford schoolchildren was similar to that noted in other series.^{4 8-14} The one-minute school screening test proved an effective method of examining for a scoliosis with only 18% of false-positives when submitted to radiographic confirmation. The radiographic screening of schoolchildren, however, raises important ethical questions, particularly in relation to the dosage to the breasts and gonads. By taking the spinal radiograph in the posteroanterior direction and incorporating an air gap the breast dose to the adolescent girl is reduced by a factor of 50, a dose which is similar to the annual background dose received in many parts of Britain.⁵ This is particularly important when radiographically monitoring those with progressive curves.

Previous screening programmes have produced high prevalence rates of scoliosis. Most cases are usually classed as idiopathic, even though very few actually progress. This study explains this finding by showing that a substantial proportion of scolioses are entirely, or partially, a result of a tilt of the sacrum in the direction of the convexity of the curve. Scoliosis did not progress in any of the children with curves caused solely by a sacral tilt. The compensatory lumbar scoliosis commonly produced by a tilt of the sacrum secondary to inequality in the legs is never progressive.³ Children with this problem present because of their unequal leg lengths, and their compensatory scoliosis is noted as an associated feature on clinical examination. Patients in this study whose scoliosis was a result of a sacral tilt would not have presented clinically because

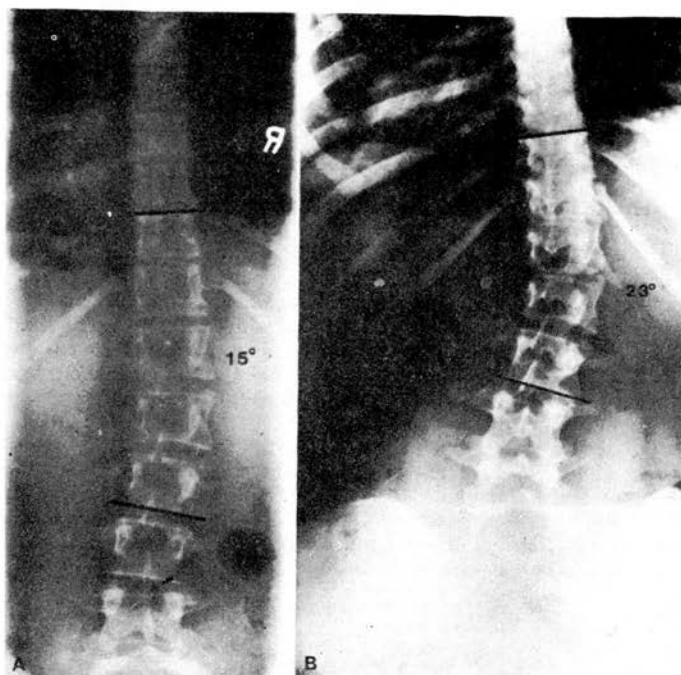


FIG 3—(a) Posteroanterior radiograph of thoracolumbar spine. There was a 15° thoracolumbar scoliosis and no sacral tilt. (b) Posteroanterior radiograph of spine of same patient one year later. Curve had progressed to 23° .

the tilt of the sacrum is a function of asymmetry of the pelvis rather than unequal leg lengths.

Before the introduction of school screening programmes analysis of patients presenting for treatment of a scoliosis showed a large preponderance of girls over boys, almost consistent progression, and relatively large numbers of thoracic curves.³ With the advent of screening, many small non-progressive lumbar curves of little importance have been detected with a female:male sex ratio approaching 1:1. Further cohort studies should include a horizontal radiographic level so that minor, non-progressive curves, as a consequence of a pelvic tilt, can be identified.

References

- ¹ Bjure J, Nachemson A. Non-treated scoliosis. *Clin Orthop* 1973;**93**:44-52.
- ² Kane WJ. An analysis of scoliosis prevalence studies. In: Zorab PA, ed. *Scoliosis: proceedings of a fifth symposium*. London: Academic Press, 1977:415-531.
- ³ James JIP. *Scoliosis*. Edinburgh: Churchill-Livingstone, 1976:7.

- ⁴ Brooks HL. Idiopathic scoliosis—results of a prospective study. In: Zorab PA, ed. *Scoliosis: proceedings of a fifth symposium*. London: Academic Press, 1977;11-30.
- ⁵ Ardran GM, Coates R, Dickson RA, Dixon-Brown A, Harding FM. Assessment of scoliosis in children. Low-dose radiographic technique. *Br J Radiol* (in press).
- ⁶ Cobb JR. Outline for the study of scoliosis. In: Edwards JW, ed. *Instructional course lecture*. Ann Arbor, Michigan: the American Academy of Orthopaedic Surgeons, 1948;5:261.
- ⁷ Whittle MW, Evans M. Instrument for measuring the Cobb angle in scoliosis. *Lancet* 1979;i:414.
- ⁸ Ottander HG. Simple x-ray method for measuring leg length discrepancy. Technical note. *Ilford x-ray focus* 1977;15:3.
- ⁹ Armstrong GWD, Adair IV, Van Wijk MR. The evaluation of Moire topography as a method for scoliosis screening programme. *J Bone Joint Surg* 1977;59B:505.
- ¹⁰ Longstein J. Screening for spinal deformities in Minnesota schools. *Clin Orthop* 1977;126:33-42.
- ¹¹ Rogala EJ, Drummonds DS. Idiopathic scoliosis a progressive study of the incidence and natural history based on a school screening of 26 447 children. *J Bone Joint Surg* 1977;59B:505.
- ¹² Shands AR, Eisenberg HB. The incidence of scoliosis in the state of Delaware. A study of 50 000 microfilms of the chest made during a survey for tuberculosis. *J Bone J Surg* 1955;37A:1243-9.
- ¹³ Span Y, Robin G, Makin K. The incidence of scoliosis in school children in Jerusalem. *J Bone Joint Surg* 1976;58B:379.
- ¹⁴ Rogala EJ, Drummond DS, Gutt J. Scoliosis incidence and natural history. *J Bone Joint Surg* 1978;60A:173-6.

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ASSESSMENT OF SCOLIOSIS IN CHILDREN - LOW DOSE RADIOGRAPHIC
TECHNIQUE.

Arday GM, Coates R, Dickson RA, Dixon-Brown A, Harding FM.

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The assessment of scoliosis in children, particularly small and possibly inconsequential curves in the community, does pose radiation risks to sensitive developing organs. Accordingly, before a screening programme could be embarked upon, a low dose radiographic technique was developed in association with radiation physicists. This was the first low dose technique to be established for spinal surgery and has been adopted in many other countries.

Assessment of scoliosis in children: low dose radiographic technique

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When scoliosis is clinically suspected in school children it may be necessary to radiograph the spine in the erect position to determine the degree of scoliosis and the presence of developmental abnormalities. The findings will be valuable to determine the future treatment of a child and to assess the necessity for operative interference. It is obviously desirable to devise a radiographic technique which will produce the information required with a low dose of radiation, bearing in mind the age of the children and the fact that subsequent radiography may be required on many occasions.

In a proposed survey in the Oxford Region on school children between the ages of nine and 14 years it was estimated that some 30000 children would undergo a physical examination together with a Moiré fringe surface topography examination (Armstrong, *et al.*, 1977). Similar surveys indicated that perhaps 2600 of these children might have minimal degrees of scoliosis in which it would be desirable to perform a single radiographic examination of the spine for future assessment. Of these 2600 children, it was then estimated that 700 would require subsequent radiographic examinations be-

cause their degree of spinal deformity would be such that progression might be expected and treatment required. When the backlog of 30000 children in this age group had been dealt with the annual numbers in future years would of course be reduced to approximately one fifth.

The technique which was proposed was an AP erect film up to 40 cm in length using approximately 90 kV, 2.5 mm Al filtration, a moving anti-scatter grid, Du Pont Quanta II intensifying screens (Cronex 4 film) with a focus-film distance of 120 cm. The skin dose for this technique was usually between 0.6-1.5 mGy (60-150 mrad).

In view of the numbers and ages of the children it was considered valuable to reduce the dose if possible.

METHOD

1. The examination was carried out in the PA projection to reduce the dose to the developing breasts to about 10%. The dose to the thyroid and that to the gonads is reduced because of the intervention of the bony parts of the spine and pelvis. This technique appears to be satisfactory at least for

Technical notes

the initial survey even if small areas of the spine have to be re-examined in more detail by other techniques. It allows determination of degrees of angulation and tilting and exclusion of the principal developmental defects.

2. In view of the success of the air-gap technique for examining chests (Ardran and Crooks, 1964; 1976) a similar technique was developed using a 15 cm air gap and a focus-film distance (FFD) of 365 cm. Removal of the anti-scatter grid resulted in a reduction of the dose by a factor of about four, and provided the beam is no wider or longer than necessary, the resultant films are adequate for the purpose.

3. Should further dose reduction be required this can be achieved by the use of a film-screen combination such as the 3M Alpha 16 with XM film or Du Pont Quanta III screens with Cronex 4 film, either of which approximately halves the dose.

RESULTS

The skin doses were measured in three groups of patients using Harshaw LiF TLD-100 chips, $3.2 \times 3.2 \times 1$ mm thick. Three chips were used in each sachet.

1. Skin doses were measured over the lower sternum of three patients examined in the AP projection, focus-film distance 120 cm using Ilford standard screens and Cronex 4 film. The doses recorded were 2.47, 3.01 and 1.42 mGy, average 2.3 mGy. These screens were not routinely used and a further three patients were examined with the same technique but using Du Pont Quanta II screens. The range here was 0.62 to 1.42 mGy, with an average of 0.96 mGy.

2. Thirty-nine patients were examined in the PA projection with the 15 cm air gap and 365 cm FFD; the kV was in the range of 80 to 102 and Quanta II screens and Cronex 4 film were used. Skin doses on the centre of the back of the patient ranged from 0.09 to 0.29 mGy with an average of 0.19 mGy.

3. In order to estimate the dose to the breast under circumstances given in 2 above, TLD's were placed just medial to the right and left nipples which was considered likely to indicate the maximum breast dose. The same TLD was used on 13 to 15

patients in order that the dose might build up to a reliable figure. The average dose (42 patients) to the breast was found to be 0.016 mGy. Similarly, TLD's were placed on the crico-thyroid region; the average dose recorded was 0.012 mGy. The dose just medial to the right nipple was usually a little higher than that on the left side, the average difference being 0.008 mGy. This was probably due to shielding of the medial aspect of the left breast by the heart.

CONCLUSIONS

The adoption of the technique recommended has resulted in reducing the radiation dose to these children by a factor of about four and the breast dose to less than 2% of what it might have been.

The radiographs produced are of adequate quality for a survey of this kind and are also suitable for routine follow-up of scoliosis patients. A further reduction in scatter could be obtained by increasing the size of the air gap up to double the distance. This would result in some enlargement unless the focus-film distance was further increased. The dose reduction brings the procedure well within the range of variations in natural annual background dose which would be received if one were to live in different parts of the country; this is considered important particularly in the light of recent evidence indicating that the developing breast is relatively radiosensitive and the statement in ICRP 26 (1977) that radiation should not be used without balancing the benefit against the risk.

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We are grateful to Dr. J. C. MacLarnon for his collaboration and to Miss S. Twemlow for radiographic help and to Miss P. Fursdon for radiographic help and technical assistance.

REFERENCES

- ARDRAN, G. M. and CROOKS, H. E., 1964. The reduction of scatter fog in chest radiography. *British Journal of Radiology*, **37**, 477-9.
- 1976. Chest Symposium—Radiography of the Chest. *X-ray Focus*, **15**, 26-34.
- ARMSTRONG, G. W. D., ADAIR, I. V. and VAN WIJK, M. R., 1977. The evaluation of Moiré topography as a method for scoliosis screening. *Journal of Bone & Joint Surgery*, **59B**, 505.
- INTERNATIONAL COMMISSION ON RADIOLOGICAL PROTECTION, 1977. Recommendations of the ICRP 26. *Annals of the ICRP*, **1**, 12, 18.

SCOLIOSIS IN THE COMMUNITY.

Dickson RA.

British Medical Journal; 286: 615-618, 1983.

A second and larger epidemiological survey of more than 5 000 school-children was performed and three types of spinal deformity clearly differentiated. Fifteen percent of the children had clinical evidence of a spinal deformity and in almost 40% this was due to a leg length inequality. Of the remainder, whose scoliosis was intrinsic to the spine, deterioration was only observed in a small minority (true idiopathic scoliosis), all other deformities remaining static or improving.

Scoliosis in the community

ROBERT A DICKSON

Abstract

Screening for scoliosis at schools has become more and more popular despite the lack of knowledge concerning the clinical course of idiopathic scoliosis. An epidemiological study of 5303 schoolchildren showed three types of scoliosis in the community: (1) pelvic tilt scoliosis (an inconsequential deformity caused by an inequality in the length of the legs but accounting for almost 40% of curves detected); (2) spinal scoliosis (a minor asymmetry of the spine in the coronal plane that tends to remain static or to resolve and which may be normal in growing children, accounting for the remaining 60%); and (3) progressive scoliosis (10% of the spinal scolioses measuring 10° or more that progress by 5° or more a year). Progressive scoliosis resembles idiopathic scoliosis because in girls with right thoracic curves the potential for progression is appreciable. Until the natural history is better established growing awareness in the community of spinal deformity should help earlier detection, and screening should be directed towards providing subjects for further epidemiological work.

Introduction

Screening for scoliosis was first performed in the early 1940s to detect paralytic spinal deformities resulting from the last poliomyelitis epidemics.¹ Attention was directed to idiopathic scoliosis 20 years later, and since then screening has mushroomed and gained worldwide enthusiasm. From the United States to Japan and Hungary to Canada screening is going on apace—in

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BMJ/82/83

some countries it is even compulsory.²⁻²⁰ By rapid crude visual examination as many as 25% of normal adolescents appear to have a scoliosis, yet only two per thousand ever achieve a curve magnitude of 20°, for which treatment is usually recommended. Interestingly, they have all been regarded as being cases of idiopathic scoliosis.

Some sense was recently injected into this problem when the Oxford Scoliosis Study Group showed that as many as 40% of spinal deformities in people in the community were minor, non-progressive, lumbar scolioses secondary to a tilt of the pelvis caused by an inequality in the length of the legs (pelvic tilt scoliosis).²¹ Of the remaining 60%, which at least appeared to be due to asymmetry of the spine (spinal scoliosis), only one fifth showed evidence of progression and therefore resembled true idiopathic scoliosis. Accordingly, a large scale epidemiological survey of more than 5000 adolescents in one community has been performed that sought to clarify what idiopathic scoliosis really is and what factors favour progression.

Patients and methods

Altogether 5303 schoolchildren (2613 girls and 2690 boys aged 10-14) were screened for a spinal deformity by an experienced senior physiotherapist who examined each child standing and leaning forward. Those with visual evidence of asymmetric trunk topography had a low dose posteroanterior spinal x ray examination.²² From these films the site of the curve—that is, the position of the apical vertebra—and the direction—that is, the side to which the convexity of the curve was directed—were recorded. The magnitude of the curve was determined using Whittle's adaptation²³ of Cobb's method.²⁴ The prevalence rate of scoliosis in the community, particularly for the two major subgroups of pelvic tilt scoliosis and spinal scoliosis, was then analysed using these variables.

The 150 children with curves measuring 10° or more were then carefully examined to exclude a concomitant musculoskeletal condition. Radiographs were taken again at six and at 12 months. The magnitude of the curve was recorded from these follow up films and progression (an increase by 5° or more) or regression (a reduction by 5° or more) determined.

Statistical analysis was by the χ^2 test, Fisher's exact test, and the binomial two-tailed test.

Results

Table I shows the prevalence rate of all scolioses and the proportions of children with spinal and pelvic tilt scoliosis according to the size of the curve. The prevalence rate of scoliosis was almost 15%, with pelvic tilt scoliosis representing almost 40% of scolioses of 5° or more. As the magnitude of the curve increased so the proportion of children with spinal scoliosis increased and the proportion with pelvic tilt scoliosis diminished reciprocally. Only two children per thousand screened had a curve magnitude of 20° or more, and only one of these

TABLE 1—*Prevalence of all scolioses and the proportions of children with spinal scoliosis (SS) and pelvic tilt scoliosis (PTS)*

Curve size (degrees)	No	Prevalence all (%)	Proportions	
			SS (%)	PTS (%)
<5	241	4.5		
5-9	377	7.1	57	43
10-14	105	2.0	69	31
15-19	32	0.6	75	25
>20	13	0.2	92	8
Total	768	14.4	61	39

had a pelvic tilt scoliosis (this was because the legs were extremely unequal in length and treatment had never been sought).

Table II shows the prevalence rate of scoliosis of 5° or more in relation to the sex of the children. The prevalence rate was significantly higher for girls for all scolioses ($p < 0.01$) and for spinal scoliosis ($p < 0.01$). Furthermore, for all scolioses and for spinal scoliosis the proportion of girls with curves of 15° or more was significantly greater than that of girls with curves measuring 5° to 9° ($p < 0.01$). The proportions of girls and boys with pelvic tilt scoliosis were not significantly different overall or at any magnitude of curve ($p > 0.05$).

Table III shows the site of the curve. Almost half of all scolioses were in the lumbar region, nearly twice as many as in the thoracic or thoracolumbar regions ($p < 0.001$). This significant preponderance of lumbar curves was lost with curves of 15° or more ($p > 0.05$). For spinal scoliosis, however, most curves were in the thoracic region ($p < 0.001$), but again significance was lost with curves of 15° or more ($p > 0.05$). The lumbar curve occurred most often in pelvic tilt scoliosis ($p < 0.001$), as would be expected in curves caused by a tilt of the pelvis.

Table IV shows the direction of the curve. Spinal scoliosis was more frequently left sided, thoracic ($p < 0.05$), thoracolumbar ($p < 0.01$), and lumbar ($p < 0.001$). The left side predominated in curves of 5°-9° in the thoracic ($p < 0.05$) and thoracolumbar regions ($p < 0.01$) but was not significant in curves of 10° or more ($p > 0.05$), whereas the left side was significant with curves of 15° or more in the lumbar region ($p < 0.05$). In pelvic tilt scoliosis, however, curves to the right and left sides were equally represented. When the direction of the curve was analysed in relation to the sex of the children there was no difference in left sided preponderance for thoracolumbar or lumbar curves. In the thoracic region, however, left sided preponderance was observed only in boys ($p < 0.01$), while in girls the proportions with right and left sided curves were not significantly different ($p > 0.05$).

No progression or regression occurred in the children with pelvic tilt scolioses. Ten per cent of spinal scolioses progressed and 18% regressed. None of the curves in a particular site progressed or regressed significantly ($p > 0.05$), nor were thoracolumbar or lumbar curves in girls or boys or on the right or left side associated with progression or regression ($p > 0.05$). For thoracic curves, however, progression tended to be associated with right sided curves and regression with left sided curves. Furthermore, the progression of right thoracic curves was significantly more common in girls than boys ($p < 0.01$).

TABLE II—Prevalence of scoliosis and sex of children

Curve size (degrees)	All			Spinal scoliosis			Pelvic tilt scoliosis		
	Girls (%)	Boys (%)	Girl:boy ratio	Girls (%)	Boys (%)	Girl:boy ratio	Girls (%)	Boys (%)	Girl:boy ratio
5-9	7.7	6.6	1.2:1	4.5	3.6	1.3:1	3.1	3.0	1.0:1
10-14	2.7	1.3	2.1:1	2.0	0.8	2.5:1	0.8	0.5	1.6:1
15-19	1.0	0.3	3.3:1	0.7	0.2	3.5:1	0.2	0.1	2.1:1
>20	0.5	0	—	0.5	0	—	0.04	0	—
Total	11.9	8.2	1.5:1	7.7	4.6	1.7:1	4.14	3.6	1.2:1

TABLE III—Site of curve

Curve size (degrees)	All			Spinal scoliosis			Pelvic tilt scoliosis		
	No of curves	Thoracic (%)	Thoracolumbar Lumbar (%)	No of curves	Thoracic (%)	Thoracolumbar Lumbar (%)	No of curves	Thoracic (%)	Thoracolumbar Lumbar (%)
5-9	394	24	26	232	40	38	162	0	9
10-14	122	30	23	89	40	26	33	0	15
15-19	34	36	32	26	35	35	8	0	25
>20	17	36	24	16	38	25	1	0	100
Total	567	25	26	363	40	34	204	0	11
									89

TABLE IV—*Direction of curve*

Curve size (degrees)	Spinal scoliosis											
	Thoracic			Thoracolumbar			Lumbar			Pelvic tilt scoliosis		
	No of curves	Right (%)	Left (%)	No of curves	Right (%)	Left (%)	No of curves	Right (%)	Left (%)	No of curves	Right (%)	Left (%)
5-9	93	38	62	89	33	67	50	28	72	162	50	50
10-14	36	47	53	23	30	70	30	43	57	33	52	48
15-19	9	33	67	9	22	78	8	—	100	8	25	75
> 20	6	67	33	4	75	25	6	—	100	1	100	—
Total	144	41	59	125	34	66	94	29	71	204	50	50

Discussion

Screening has been defined as "the presumptive identification of unrecognised disease or defect by the application of tests, examinations, or other procedures which can be applied rapidly."²⁵ Screening tests sort out apparently well people who have a disease from those who probably do not, and a prerequisite for this is that the clinical course of the condition should be known.²⁶ It is extraordinary that hundreds of thousands of children throughout the world have been subjected to a crude visual examination of the spine to identify asymmetry, which is supposed to be idiopathic scoliosis, whatever that condition is and however it behaves. When as many as a quarter of apparently normal children have visual evidence of spinal asymmetry in the coronal plane by this crude test then substantially more—possibly all children—would show evidence of asymmetry with a more sensitive test. This supports the view of anatomists two centuries ago that there is a scoliosis in every spine, albeit of small magnitude.^{27 28} Indeed, a scoliosis surgeon, given an x ray film and a protractor, could find a scoliosis somewhere in any spine. When likened to screening for breast cancer we are screening for the presence of the breasts as it were, and there is nothing on examination of the spine akin to the underlying pathological breast mass. That almost 15% of normal children in this study had spinal asymmetry yet only two per thousand had a severe curve clearly shows that other important factors superimposed on a lateral curvature of the spine make it an idiopathic scoliosis.

An important diluting factor is the presence of pelvic tilt scoliosis due to an inequality in the length of the legs, which accounts for almost 40% of curves detected and which is manifestly not idiopathic scoliosis. The even female to male sex ratio, the diminishing prevalence with the magnitude of the curve, and the site being always low down in the spine and favouring no particular direction not increasing in size account for the high proportion of irrelevant lumbar curves that so typifies "schooliosis." The hitherto unrecognised contribution of pelvic tilt scoliosis has made interpreting the results of other screening studies extremely difficult, helped by a lack of awareness of the definition of common epidemiological terms, "incidence" and "prevalence" appearing to be freely interchangeable words of the same meaning. The high prevalence rate of pelvic tilt scoliosis is also important in relation to radiation dosage. In screening programmes any child with visual evidence of spinal asymmetry promptly undergoes radiography by conventional techniques. Thus far only two groups have sought to protect these children by reducing radiation dosage to a minimum.^{22 29}

When pelvic tilt scoliosis has been identified and then eliminated attention may be directed towards scoliosis that is at least inherent to the spine—spinal scoliosis—where the characteristics of some curves begin to resemble idiopathic

scoliosis. Girls predominate, particularly among children with bigger curves, and the thoracic region is the commonest site. Even so, although there are more left sided curves in the lumbar region in both "schooliosis" and idiopathic scoliosis, there are still more left thoracic curves in people in the community in contradistinction to idiopathic scoliosis. Ninety per cent of spinal scolioses either remain static or regress. The 10% that progress resemble true idiopathic scoliosis, with right thoracic curves in girls showing a real potential for progression.

Thus there seem to be three types of scoliosis in the absence of any musculoskeletal disease or congenital spinal anomaly. Pelvic tilt scoliosis is due to an inequality in the length of the legs. Spinal scoliosis probably reflects irrelevant coronal plane asymmetry of the spine in normal healthy children. Idiopathic scoliosis is by far the least prevalent but by far the most important. It is the 10% of spinal scoliosis that is progressive, and thus right thoracic curves in girls demand the closest scrutiny. Clearly other factors are important, and recent evidence suggests that while asymmetry of the spine in the coronal plane is so common as to be normal, asymmetry of the spine in the median sagittal plane in the nature of a lordosis at the apex of the curve is the essential lesion of idiopathic scoliosis.³⁰ As much attention should therefore be directed towards viewing the spine from the side as from the front or back.

While it is disturbing that many children with progressive scoliosis go untreated because they are not detected³¹ and that the magnitude of the curve of those who do present clinically is often too great to allow conservative treatment,³² it is clear that screening at school for scoliosis as it is practised does not primarily detect idiopathic scoliosis. It is an enormous waste of time, energy, and money of an already overstretched establishment, not to mention the hazards of taking radiographs of vast numbers of children who do not have idiopathic scoliosis for the benefit of the small number who do. If the results of the many reported studies had focused more on clinical course, and therefore been true epidemiological surveys we would have been much nearer having a prerequisite for a screening programme. Fortunately, where screening has been performed the magnitude of the curve in children at presentation has noticeably dropped, so that more children may have the benefit of effective conservative treatment.³³ This has not been due to the screening directly, since only small numbers of the population at risk have been examined, but is due to greater awareness in the community in general of scoliosis, and this is where resources should be directed until more is known about this complex condition to which the term idiopathic is singularly appropriate.

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References

- ¹ Lonstein JE. Screening for spinal deformities in Minnesota schools. *Clin Orthop* 1977;**126**:33-42.
- ² Kane WJ, Moe JH. A scoliosis-prevalence survey in Minnesota. *Clin Orthop* 1970;**69**:216-8.
- ³ Strayer LM. The incidence of scoliosis in the post-partum female on Cape Cod. *J Bone Joint Surg* 1973;**55A**:436.
- ⁴ Sells CJ, May EA. Scoliosis screening in public schools. *Am J Nurs* 1974;**74**:60-2.
- ⁵ Segil CM. The incidence of scoliosis in the Bantu and white population groups in Johannesburg. *J Bone Joint Surg* 1974;**56B**:393.
- ⁶ Golomb M, Taylor TF. Screening adolescent schoolchildren for scoliosis. *Med J Aust* 1975;**2**:761-2.
- ⁷ Brooks HL, Azen SP, Gerberg E, et al. Scoliosis: a prospective epidemiological study. *J Bone Joint Surg* 1975;**57A**:968-72.
- ⁸ Span Y, Robin G, Makin M. The incidence of scoliosis in schoolchildren in Jerusalem. *J Bone Joint Surg* 1976;**58B**:379.
- ⁹ Lonstein JE, Winter RB, Moe JH, et al. School screening for the early detection of spine deformities. *Minn Med* 1976;**59**:51-7.
- ¹⁰ Inoue S. Moire topography for the early detection of scoliosis and evaluation after surgery. *Orthopaedic Transactions* 1978;**2**:76.
- ¹¹ Abbott EV. Screening for scoliosis: a worthwhile preventive measure. *Can J Public Health* 1977;**68**:22-5.
- ¹² Flynn JC, Riddick MF, Keller TL. Screening for scoliosis in Florida schools. *J Fla Med Assoc* 1977;**64**:159-61.
- ¹³ Newman DC, DeWald RL. School screening for scoliosis. *IMJ* 1977;**152**:31-4.
- ¹⁴ Bellyei A, Czeizel A, Barta O, Magda T, Molnar L. Prevalence of adolescent idiopathic scoliosis in Hungary. *Acta Orthop Scand* 1977;**48**:177-80.
- ¹⁵ Ascani E, Salsano V, Giglio G. The incidence and early detection of spinal deformities. *Ital J Orthop Traumatol* 1977;**3**:111-7.
- ¹⁶ O'Brien JP, van Akkerveeken PF. School screening for scoliosis. Results of a pilot study. *Practitioner* 1977;**219**:739-42.
- ¹⁷ Adair IV, Van Wijk MC, Armstrong GWD. Moire topography in scoliosis screening. *Clin Orthop* 1977;**129**:165-71.
- ¹⁸ Rogala EG, Drummond DS, Gurr J. Scoliosis: incidence and natural history. *J Bone Joint Surg* 1978;**60A**:173-6.
- ¹⁹ Smyrnis PN, Valavanis J, Alexopoulos A, Siderakis G, Giannestras NJ. School screening for scoliosis in Athens. *J Bone Joint Surg* 1979;**61B**:215-7.
- ²⁰ Burwell RG, James NJ, Johnson F, Webb JK. The rib hump score: a guide to referral and prognosis? *J Bone Joint Surg* 1982;**64B**:248.
- ²¹ Dickson RA, Stamper P, Sharp AM, Harker P. School screening for scoliosis: cohort study of clinical course. *Br Med J* 1980;**281**:265-7.
- ²² Ardran GM, Coates R, Dickson RA, Dixon-Brown A, Harding FM. Assessment of scoliosis in children. Low-dose radiographic technique. *Br J Radiol* 1980;**53**:146-7.
- ²³ Whittle MW, Evans M. Instrument for measuring the Cobb angle in scoliosis. *Lancet* 1979;**i**:414.
- ²⁴ Cobb JR. Outline for the study of scoliosis. In: Edwards JW, ed. *Instructional course lecture*. Vol 5. Ann Arbor, Michigan: American Academy of Orthopaedic Surgeons, 1948:261.
- ²⁵ Commission on Chronic Illness. *Chronic illness in the United States*. Vol I. Cambridge, Massachusetts: Harvard University Press, 1957.
- ²⁶ Whitby LG. Screening for disease. Definitions and criteria. *Lancet* 1974;**ii**:819-22.
- ²⁷ Sabatier. *Traite complete d'anatomie*. Paris: 1777. Quoted by A Farkas.²⁸
- ²⁸ Farkas A. Physiological scoliosis. *J Bone Joint Surg* 1941;**23**:607-27.
- ²⁹ De Smet AA, Fritz SL, Asher MA. A method for minimising the radiation exposure from scoliosis radiographs. *J Bone Joint Surg* 1981;**63A**:156-8.
- ³⁰ Dickson RA, Lawton JO, Archer IA, et al. Combined median and coronal plane asymmetry—the essential lesion of progressive idiopathic scoliosis. *J Bone Joint Surg* (in press).

- ³¹ Nachemson A. A long-term follow-up study of non-treated scoliosis. *Acta Orthop Scand* 1968;**39**:466-76.
- ³² Belstead JS, Edgar MA. Early detection of scoliosis. *Br Med J* 1978;ii: 937-8.
- ³³ Torell G, Nordwall A, Nachemson A. The changing pattern of scoliosis treatment due to effective screening. *J Bone Joint Surg* 1981;**63A**:337-41.

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SCHOOL SCREENING AND PELVIC TILT SCOLIOSIS.

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Lancet; ii: 152-154, 1984

Children with pelvic tilt scoliosis in the epidemiological survey of more than 5 000 school-children (Publication 3) were studied and it was observed that this type of scoliosis could arise from asymmetry of the pelvis as well as a leg length inequality. This type of spinal deformity can only be reliably differentiated from idiopathic scoliosis by the incorporation of a horizontal radio-opaque fluid level on the initial low dose spinal films.

TABLE II—INCIDENCE OF KERATOMALACIA (PER 1000 PRESCHOOL CHILDREN) IN THE STUDY AREAS

Area	Year of study				
	1st	2nd	3rd	4th	5th
A	0.47	0.32	0.07	0.11	0.10
B	0.57	0.52	0.57	0.09	0.08
C	0.80	0.73	0.60	0.67	0.60

Area A: Programme implemented from 1st year; B: programme implemented from middle of 3rd year; C: non-programme area.

TABLE III—ASSOCIATION BETWEEN KERATOMALACIA AND RECEIPT OF MASSIVE DOSE OF VITAMIN A

Dose of Vit A	Cases (keratomalacia)	Controls (without keratomalacia)	Total
Not received	30 (94%)	54 (55%)	84 (64%)
Received	2 (6%)	45 (45%)	47 (36%)
Total	32 (100%)	99 (100%)	131 (100%)

$$\text{Odds ratio (R)} = \frac{45 \times 30}{54 \times 2} = 12.5$$

$$\% \text{ effectiveness of dose} = \frac{100(R-1)}{R} = \frac{100 \times 11.5}{12.5} = 92$$

Only children from areas A and B after the programme was initiated are included.

of children. Whenever necessary symptomatic treatment was given, and the children recovered completely within 24 h.

In general about 60% of the patients with keratomalacia attending the hospitals were from the slum areas and the rest were from non-slum areas. In the first year the proportion of cases that came from areas A and B was about 19%, and this declined to about 5% at the end of the study (table I), whereas in non-programme slum areas it increased slightly from about 17 to 29%.

The incidence of keratomalacia in areas A and B decreased over the years. In area A, where the programme was in operation at the start of our study, the incidence decreased from 0.47 in the first year to 0.10 in the fifth year; in area B, where the programme was initiated in the middle of the 3rd year, it decreased from 0.57 to 0.08. In area C the incidence fell only slightly from 0.80 to 0.67 (table II).

Of the patients with keratomalacia only 6% reported having received vitamin A, whereas 45% of the controls had received the concentrate (table III). The odds ratio was 12.5, indicating that keratomalacia was more likely in children who had not received vitamin A. The percent effectiveness of the supplement was 92%.

DISCUSSION

Until now, the question of whether distribution of massive doses of vitamin A will reduce the incidence of keratomalacia in preschool children has not been answered. The evidence in the present study clearly indicates that large supplements of vitamin A given six monthly can do so. A conventional intervention trial comparing prevalence in the community before supplementation with prevalence after supplementation was not possible because of the very low community prevalence of corneal xerophthalmia. Hence our use of a case-control approach, which is good for estimating relative risk and which performs with more power when applied to a high-risk population. The incidence of keratomalacia in the study areas before institution of the

programme of 0.7 to 0.8 per 1000 indicates that keratomalacia is a public-health problem there. The WHO prevalence criterion for corneal involvement indicative of significant vitamin A deficiency among the at-risk group of children is 0.01%.³ In view of the large number of preschool children at risk of corneal xerophthalmia, the need for an effective prophylaxis programme is obvious. The half yearly distribution of vitamin A reduced the incidence of keratomalacia by about 80%. In slum areas where the programme had not been implemented the decrease was of the order of only 20%. In an earlier field trial which tested the impact of massive doses of vitamin A on conjunctival signs, there was about 75% reduction in the prevalence.⁴ The present study showed a pronounced association between the receipt of massive-dose supplements and absence of keratomalacia—the high odds ratio (12.5) clearly indicated that keratomalacia was more likely to occur in children who had not received the supplements. Studies done in Indonesia have also indicated that if all children received 200 000 IU of vitamin A every six months most nutritional blindness in the country would be prevented.⁶ A mass distribution programme in El Salvador did not reduce the number of cases of corneal xerophthalmia presenting to clinics and hospitals,⁸ perhaps because the hospital statistics do not represent what is happening in the community, or perhaps because children at greatest risk were missed by the distribution mechanism.⁹ In our study we took steps to ensure that the supplements were distributed to a large proportion (87%) of the eligible children.

Our study seems to confirm WHO's expectations:³ "Although the goal of controlling florid forms of severe malnutrition like kwashiorkor, marasmus and keratomalacia by the end of the century may be difficult to attain, it is not unrealistic as far as keratomalacia is concerned, provided the knowledge now in hand is applied". The simple measure of distributing massive doses of vitamin A can reduce nutritional blindness if steps are taken in developing countries to ensure that all children at high risk actually receive the vitamin regularly. It has been reported that the massive-dose vitamin A programme in India is poorly implemented in many states. Our demonstration of its effectiveness in preventing nutritional blindness makes it necessary to improve its implementation.

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REFERENCES

- Editorial. Xerophthalmia control. *Lancet* 1982; ii: 28–29.
- Sommer A, Hussain G, Tarwotjo I, Susanto D, Soegiharto T. Incidence, prevalence, and scale of blinding malnutrition. *Lancet* 1982; i: 1407–08.
- Control of vitamin A deficiency and xerophthalmia: WHO Tech Rep Ser, no 672. Geneva: WHO, 1982.
- Swaminathan MC, Susheela TP, Thimmayamma BVS. Field prophylactic trial with a single massive oral dose of vitamin A. *Am J Clin Nutr* 1970; 23: 119–22.
- Vijayaraghavan K, Rao NP. An evaluation of the national prophylaxis programme against blindness due to vitamin A deficiency. *Nutr Rep Int* 1982; 25: 431–41.
- Nutrition Blindness Prevention Project, Indonesia. Characterization of vitamin A deficiency and xerophthalmia and the design of effective intervention program. New York: Helen Keller International Ltd, 1980: 5.
- Milton RC. Evaluation of the efficacy of programs for the control of severe xerophthalmia. *Am J Clin Nutr* 1982; 35: 140–45.
- Sommer A. Assessment of xerophthalmia and the mass vitamin A program in El Salvador (Sept 1973–Dec 1974). *J Trop Ped Env Child Hlth* 1976; 22: 136–48.
- Sommer A. Nutritional blindness, xerophthalmia and keratomalacia. New York: Oxford University Press, 1981.

Screening for Disease

SCHOOL SCREENING AND PELVIC TILT SCOLIOSIS

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Summary 5303 schoolchildren aged 10–14 years were screened for scoliosis in an epidemiological survey. 375 (7.1%) had curves of 5–9° inclusive and of these 138 had scoliosis secondary to a tilt of the pelvis. Radiographic measurements showed that the pelvic tilt was due to pelvic asymmetry, leg length inequality, or both, but bore no relation to the height of the iliac crests. Pelvic asymmetry occurred more commonly in combination with leg length inequality than as an isolated finding. In order to detect idiopathic scoliosis in the thoracolumbar or lumbar region, pelvic tilt scoliosis must be identified and excluded, but this can only be done radiographically.

INTRODUCTION

SCHOOL screening programmes have provided limited information on the natural history of idiopathic scoliosis.^{1–3} Up to 25% of normal adolescents have clinical evidence of a spinal deformity in the forward bending position.⁴ However, more than 40% of the deformities so detected are secondary to the presence of a pelvic tilt.⁵ These small, non-progressive, predominantly lumbar curves occur with equal frequency in both sexes and in both directions.⁶ A pilot study showed that pelvic asymmetry, leg length inequality, or both in combination were the cause of the pelvic tilt.⁷ In the study reported here the radiographs of a much larger group of children with pelvic tilt scoliosis were analysed to categorise the factors producing this form of scoliosis in a large population.

SUBJECTS AND METHODS

5303 schoolchildren aged 10–14 years (2613 girls and 2690 boys) were screened for spinal deformity; all were examined standing and leaning forward. Those with visual evidence of asymmetric trunk topography had a low-dose posterior-anterior spinal radiographic examination.⁷ To minimise postural variation, one of us (R. A. D.) was present when every radiograph was taken. The patient's knees were fully extended and the feet placed together. A contrast-medium spirit level was placed on the X-ray cassette just below the femoral heads to produce a constant horizontal radiographic level.⁸ From these films the Cobb angle of the scoliosis was measured.⁹ The films of the 375 children with scoliotic curves of 5–9° were studied with respect to the inclination of the pelvis. The angle subtended by the upper border of the sacrum and the horizontal reference level indicated the degree of pelvic tilt. The angle subtended by a tangential line across the femoral heads and the horizontal reference level indicated the degree of leg length inequality (hip tilt). The angle subtended by a tangential line across the highest points of the iliac crest and the horizontal reference level was recorded to determine whether this angle was related to the degree of pelvic tilt.

The site, direction, and lower end-vertebra of each curve were also recorded. For a curve to be classified as a pelvic tilt scoliosis the lower end-vertebra had to be the upper border of the sacrum, its convexity had to be towards the low side of the pelvis, and its magnitude not greater than twice that of the degree of pelvic tilt (accepting not >1° error in measurement).¹⁰ If there was no hip tilt, the scoliosis was attributed solely to pelvic asymmetry (fig 1). If the hip tilt was the same size as the pelvic tilt, the scoliosis was

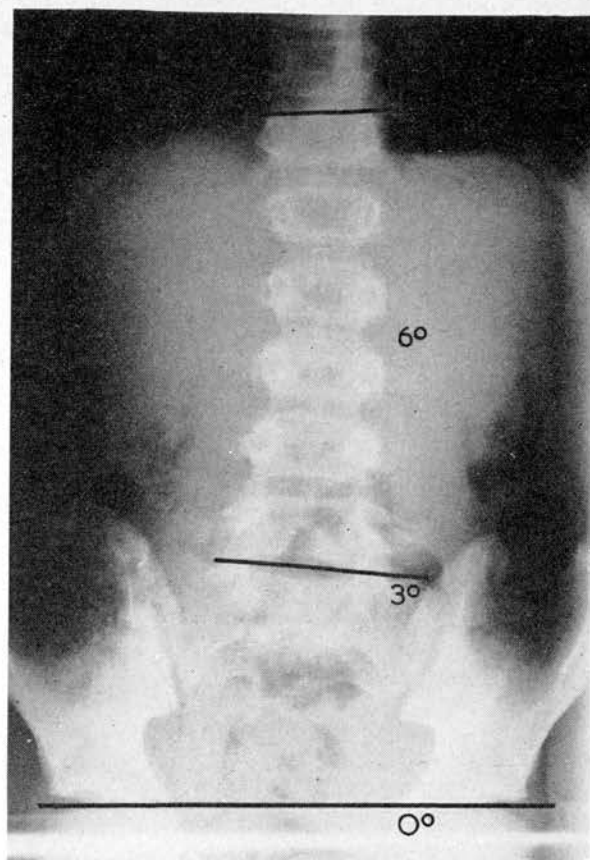


Fig 1—Pelvic asymmetry only.

attributed solely to leg length inequality (fig 2). If the hip tilt was less than the pelvic tilt, both leg length inequality and pelvic asymmetry were considered to be present (fig 3). The significance of the results was determined with the chi-square test and by calculation of the correlation coefficient.

RESULTS

138 (37%) of the 375 children with scoliotic curves of 5–9° had pelvic tilt scoliosis. Their mean age was 12.25 years. 10 radiographs were not of sufficient quality to allow analysis of the pelvis. Of the remaining 128 pelvic tilt scolioses, 63% were combined pelvic asymmetry and leg length inequality, 23% leg length inequality only, and 14% pelvic asymmetry only (see table). The proportions of boys and girls were not significantly different overall or in the three groups. 87% of curves were lumbar and 13% thoracolumbar and their proportions were not significantly different in the three groups. The proportions of left-sided curves (55%) and right-sided curves (45%) were not significantly different overall, but right-sided curves were significantly more common in the leg length inequality group ($p < 0.02$) and significantly less common in the combined group ($p < 0.02$). The height of the iliac crests bore no relation to the magnitude of pelvic tilt.

DISCUSSION

While the earlier detection of children with idiopathic scoliosis remains a priority of health care, whether this should necessarily be achieved by routine screening has been questioned.^{6,11} An important cause of the uncertainty is the screening method used (forward bend test) and whether there really is a satisfactory alternative. It has become increasingly clear that the keener the search the higher the prevalence rate

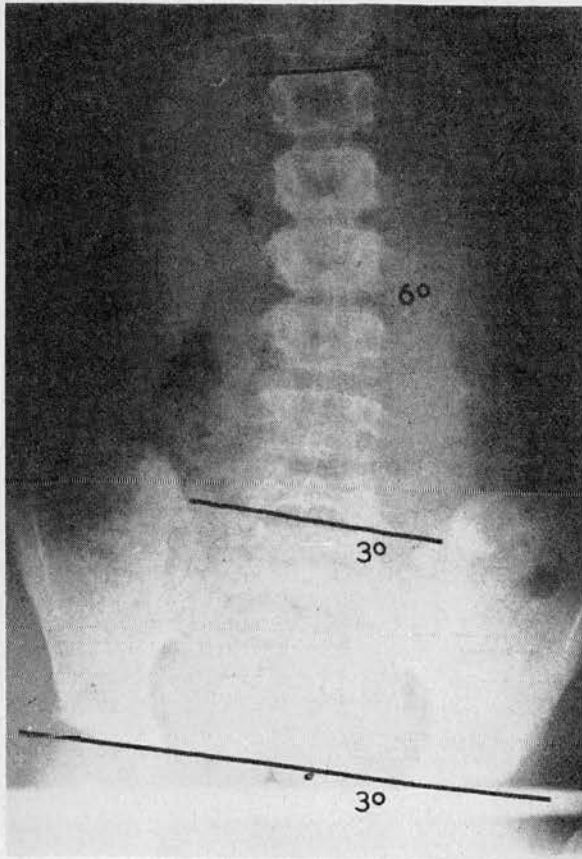


Fig 2—Leg length inequality only.

of trunk asymmetry,⁴ but whether the trunk asymmetry relates to the axial skeleton is only conjecture. Indeed, if screening is carried out by X-ray, two-thirds of girls can be shown to have mild scoliosis.¹²

When a child with idiopathic scoliosis bends forward the rotational prominence is exaggerated as the primary lordosis rotates to the side to avoid compression.^{13,14} However, when the normal lumbar lordosis flexes forward above a tilted pelvis a small loin hump is produced by the same mechanism; thus almost 50% of mild humps detected in the community have nothing to do with idiopathic scoliosis.⁶ It is entirely reasonable to envisage that the tilted pelvis is due to a minor degree of leg length inequality, but this is true in less than a quarter of such cases. The great majority are solely or partly related to pelvic asymmetry and, therefore, a sitting forward bend test would filter out only a small minority. No help is obtained from the sex of the child or a clinical estimate of pelvic tilt from the height of the iliac crests. Although there are right/left differences between the leg length inequality and combined groups there is no help from side of curve in the pelvic tilt cases as a whole. The only reliable method of sifting out pelvic tilt scoliosis is by X-ray and, even if a low-dose technique is used,⁷ this unimportant disorder does not demand this level of diagnosis.

PELVIC TILT SCLIOSIS

	Left	Right	Total (%)
Pelvic asymmetry	10	8	18 (14)
Leg length inequality	8	21	29 (23)
Combined	52	29	81 (63)
Total	70	58	128

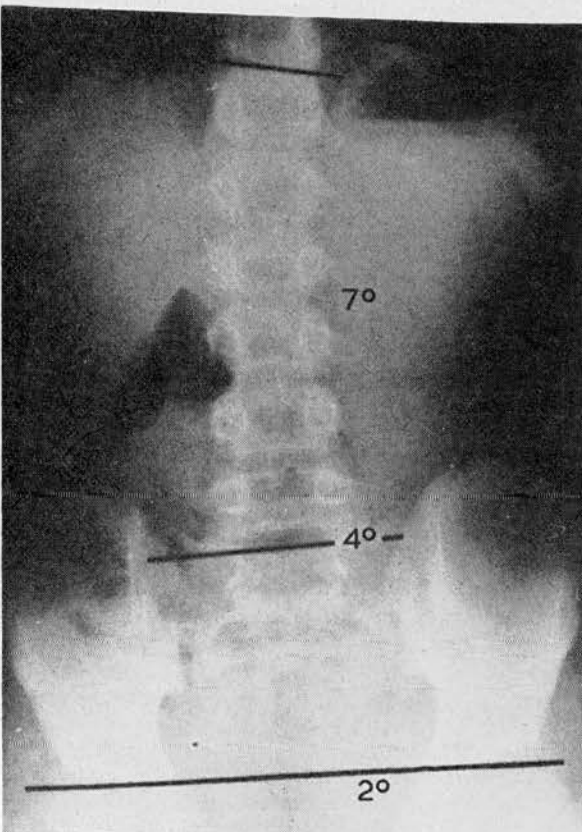


Fig 3—Pelvic asymmetry plus leg length inequality.

Frustratingly, children with idiopathic scoliosis start with straight, or nearly straight, spines and must therefore be in the screening harvest somewhere, but their early identification by means of forward spinal flexion is so diluted by children with non progressing curves as to seem hardly worthwhile. Vercauteren has recently studied trunk asymmetry in relation to the X ray appearances of the spine, and many indices of asymmetry previously thought to be important (shoulder height, scapular position, spinal balance) are quite irrelevant.¹⁵ Only a rotational prominence of ≥ 1 cm and asymmetry of the waist triangles are significantly correlated with spinal shape. Therefore a 1 cm hump might be a more sensitive index of the presence of a potentially progressive curve and would reduce the harvest somewhat. To this might be added girls with thoracic humps, who definitely have a greater likelihood of progression.⁶

Perhaps the most serious matter is what is done about those curves not due to a pelvic tilt when they are found. It has become clear that conservative treatment does not alter the course of the idiopathic deformity.¹¹ Presumably, such children must all be repeatedly observed so that the very few in whom the scoliosis progresses can have the benefit of operative treatment before the deformity becomes unacceptable. Fortunately, where screening of thousands of children is carried out routinely conservative and operative treatments are being prescribed much less frequently,¹⁶ suggesting that idiopathic scoliosis is pursuing a more benign course spontaneously.

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Clinical Sign

PRODROMAL ITCHING IN CHILDHOOD ASTHMA

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Summary In a prospective study of 79 children with asthma, 26 had prodromal itching 1–30 min before the attack, and 17 had prodromal itching during the early part of the attack. The sensation lasted up to about 30 min. Scratch marks were seen in 5 patients. In 7 patients the itching was known to the child but had not been recognised by the parents. The site of itching was constant for each patient, being the anterior part of the neck in 14 patients and the upper part of the back in 9 patients. The pathogenesis of prodromal itching is obscure, but its recognition may be of diagnostic use when taking a history and may enable an attack of asthma to be aborted or treated more promptly.

INTRODUCTION

PATIENTS with asthma sometimes experience a sensation of itching just before an attack. The site of itching is localised, being usually at the front of the neck or over the upper part of the back, and is usually constant for each patient. The patient's response is nearly always to scratch. Prodromal itching, although often experienced by patients with asthma, is unfamiliar to most doctors. We have only been able to trace a single study of prodromal itching in asthma, and this excluded young children.¹ The present study was undertaken to examine the frequency of prodromal itching in children with asthma.

PATIENTS AND METHODS

Children with asthma under the care of Booth Hall Children's Hospital were studied prospectively. Only children who had had at least 1 attack severe enough to require admission to hospital were studied. The parent(s) and children were interviewed, and a standard questionnaire was completed for each patient. Age, duration of asthma, number of admissions to hospital because of asthma, type of prophylactic treatment, use of regular oral antihistamines, and the presence of eczema were recorded. The parents and patients were asked whether itching or scratching was noticed, for how long it lasted, exactly where it was located, and whether it occurred before or during asthma attack. Prodromal itching was defined as an itching sensation (felt by the patient), or scratching (observed by the relatives), occurring just before, or during, the early part of, an attack.

RESULTS

79 patients, 53 boys and 26 girls, were studied. They were aged from 2 to 16 years (mean 7.75 ± 3.62). 27 patients (34%) had atopic eczema. The mean number of admissions to hospital because of asthma attack was 5.81. 71 patients received regular prophylactic treatment, which consisted of oral or inhaled beta₂-stimulants in 48 patients, oral slow-release theophylline preparations in 9, inhaled disodium cromoglycate in 16, inhaled corticosteroids in 28, intramuscular corticotropin in 2, and oral prednisolone in 1. 10 patients received antihistamines nightly as part of the treatment for atopic eczema. The mean length of time since diagnosis of asthma was 2.52 years.

Prodromal itching was reported in 32 patients (41%). In 7 of these, the itching sensation was known to the child but had not been recognised by the parents. In 1 case neither mother nor child (aged 7) had noticed itching, but a 13-year-old sister sharing the patient's bedroom said that she had often noticed her sister rubbing her neck just before an attack. There was no statistically significant difference between the age distribution of those with or without prodromal itching. 23 (72%) of the 32 patients with prodromal itching and 30 (64%) of the 47 without prodromal itching were boys. The excess of boys in the former group was not significant. The occurrence of prodromal itching was unrelated to any particular prophylactic treatment or antihistamine use. The frequency of eczema was the same in those with or without prodromal itching. In 4 patients, recognition of prodromal itching made it possible to abort some attacks of asthma by the early use of bronchodilators.

Prodromal itching occurred in a constant location in each patient, but in some patients it was seen in more than 1 site. The most common site was the anterior neck in the midline (14 patients), followed by the upper part of the back in the midline (9), the upper sternal area (4), the vertex (3), the side of the neck (2), the umbilical area (2), the epigastrium (1), the back of the neck (1), the left shoulder and left upper arm (1), the right side of the cheek and both legs (1), and the left side of the cheek (1).

26 of the 32 patients had itching before the attack—in 22 it began 1–2 min, and in 4, 10–30 min, before wheezing and coughing. In 17 patients, the itching was noticed during the early part of the attack itself. Many parents and children had difficulty in being precise about the duration of itching, but 10 said it lasted less than 5 min, 1 said it lasted 5–10 min, and 4 said it lasted 30 min or more. 5 said that it lasted until treatment had begun to work, and 3 said that it stopped as soon as wheezing or coughing started. Scratch marks were not systematically sought but were noticed in 5 patients with prodromal itching; in each, the scratch marks were over the upper part of the back.

A. P. WALKER AND R. A. DICKSON: REFERENCES

- Brooks HL, Azen SP, Gerberg E, Brooks R, Chau L. Scoliosis: a prospective epidemiological study. *J Bone Joint Surg* 1975; **57A**: 968–72.
- Lonstein JE. Screening for spinal deformities in Minnesota schools. *Clin Orthop* 1977; **126**: 33–42.
- Rogala EG, Drummond DS, Gurr J. Scoliosis: incidence and natural history. *J Bone Joint Surg* 1978; **60A**: 173–76.
- Burwell RG, James NJ, Johnson F, Webb JK, Wilson YG. Standardised trunk asymmetry scores. A study of back contour in healthy schoolchildren. *J Bone Joint Surg* 1983; **65B**: 452–63.
- Dickson RA, Stamper P, Sharp AM, Harker P. School screening for scoliosis: cohort study of clinical course. *Br Med J* 1980; **281**: 265–67.
- Dickson RA. Scoliosis in the community. *Br Med J* 1983; **286**: 615–18.
- Adran GM, Coates R, Dickson RA, Dixon-Brown A, Harding FM. Assessment of scoliosis in children. Low-dose radiographic technique. *Br J Radiol* 1980; **53**: 146–47.
- Ottander HG. Simple x-ray method for measuring leg length discrepancy. Technical Note. *Ilford X-ray Focus* 1977; **15**: 3.
- Whittle MW, Evans M. Instrument for measuring the Cobb angle in scoliosis. *Lancet* 1979; **i**: 414.
- Papioannou T, Stokes I, Kenwright J. Scoliosis associated with limb length inequality. *J Bone Joint Surg* 1982; **64A**: 59–62.
- British Orthopaedic Association and British Scoliosis Society. School screening for scoliosis. *Br Med J* 1983; **287**: 963–64.
- Avikainen VJ, Vaherto II. A high incidence of spinal curvature. A study of 100 young female students. *Acta Orthop Scand* 1983; **54**: 267–73.
- Adams W. Lectures on the pathology and treatment of lateral and other forms of curvature of the spine. London: Churchill, 1865.
- Dickson RA, Lawton JO, Archer JA, et al. Combined median and coronal plane asymmetry—the essential lesion of progressive idiopathic scoliosis. *J Bone Joint Surg* 1983; **65B**: 368.
- Vercauteren M, Van Beneden M, Verlaet B, Croene P, Uyttendaele D, Verdonk R. Trunk asymmetries in a Belgian school population. *Spine* 1982; **7**: 555–62.
- Lonstein J, Bjorklund S, Wanninger MH, Nelson RP. Voluntary school screening for scoliosis in Minnesota. *J Bone Joint Surg* 1982; **64A**: 481–88.

SCREENING FOR SCOLIOSIS.

Dickson RA.

British Medical Journal; 289: 269-270, 1984.

This invited editorial summarised knowledge concerning screening for scoliosis and urged for a better understanding of the natural history of spinal deformities by way of epidemiological surveys rather than screening for clinical reasons.

Screening for scoliosis

Teenagers are still being seen with idiopathic scoliosis, bent double at presentation and with such severe respiratory dysfunction as to preclude surgery¹—apparently lending powerful support to those who have enthusiastically introduced school screening programmes to detect such cases at an earlier stage.^{2,5} Such distressing tragedies evoke powerful emotions, but is the argument so simple, and is screening a suitable solution?

The cause of idiopathic scoliosis is unknown, but the pathogenesis of the three dimensional deformity has been established.⁶⁻¹¹ The typical idiopathic deformity, a lateral curvature with rotation, is secondary to a primary deformity in the sagittal plane. There is lordosis in the thoracic region, where kyphosis should exist, and the deformity is the opposite of Scheuermann's kyphosis.¹⁰ A lordosis is rotationally unstable as its axis lies posterior to the vertebral bodies. Therefore, all scolioses with rotation are lordoscolioses and not kyphoscolioses.

Recognition of the presence of the lordosis is crucial to treatment. Just as Scheuermann's kyphosis needs extension to improve it—and this is most effective, as the deformity is rotationally stable¹²—so the idiopathic lordosis might appear to need flexion. Unfortunately, on flexion the lordosis rotates to the side to produce the scoliosis (just as a piece of paper twists on being flexed towards one of its edges).⁸ The deformity cannot, therefore, be treated conservatively.¹³ What braces do primarily is to prevent flexion, though some temporary improvement may occur if reduction of the lumbar lordosis below produces thoracic extension above.¹⁰ Even if bracing were effective it would have to be continued until spinal maturity,^{14,16} which occurs 10 years after general skeletal maturity.^{17,18} Curves in the spine tend to get worse until this time, but an unusual degree of compliance would be required for a 10 year old to wear a brace for 15 years.¹⁹

If, then, conservative treatment is of little value is surgical treatment much better? The crucial factor is the potential for progression of the deformity. The traditional operation—posterior Harrington instrumentation and fusion—is addressed to the secondary scoliotic deformity. At best the only correction it gives is around 50% of the component in the coronal plane; it has no effect on the rotational prominence with which every patient presents.^{20,23} From the patient's viewpoint a lot of surgery seems to produce little gain. Moreover, the underlying lordosis implies that the back of the spine is too short, and a posterior fusion may

add insult to injury. Thus posterior surgery may indeed prevent progression in patients with the more benign, late onset variety of scoliosis, but for those with early onset with much more growth ahead quite the opposite may happen—surgery may facilitate accelerated progression.^{8,10} With bigger curves the outlook is even more gloomy. Preoperative traction of any kind has no corrective effect,^{24,25} and the surgical attack needs to be more aggressive.^{26,27} On the brighter side, newer forms of instrumentation are more promising,^{28,29} provided that attention is directed to the sagittal plane.¹⁰

The key to untreated scoliosis is the age of onset. The high morbidity and mortality rates from cardiopulmonary compromise quoted as strong arguments by screeners³⁰ are relevant only to patients with an early onset, when chest deformity may be present at the time the pulmonary parenchyma is developing.³¹ Late onset deformities, by contrast, even if they become severe, are not associated with any substantial reduction in chest function,^{30,32,33}—though patients with this type of scoliosis may fare less well socially³⁴ and psychologically.³⁵

In practice, while the serious organic problems tend to occur in patients with early onset disease most screening has been done in adolescence. The data are difficult to interpret because of differences in terminology.³⁶ Most screening has relied on a quick visual examination, when about 15% of adolescents show evidence of asymmetry of the trunk.³⁷ When the surface profile of the back is measured the rate of asymmetry rises to about 25%.³⁸ When adolescents are x rayed almost all may be shown to have a measurable scoliosis, but in half this is secondary to a tilted pelvis.³⁷ About 2% of children screened have a curve measuring 10° or more, and 0.2-0.5% have a curve measuring 20° or more.^{2,4,37} The few longitudinal studies show that scoliosis due to pelvic tilt has no potential for progression, and that while 10% of the remainder may deteriorate twice as many improve—and the rest stay the same.^{2,4,37,39}

True idiopathic scoliosis is, then, probably made up from the small fraction that progress. Girls with right thoracic curves show the greatest progression potential.^{2,4,37}

The benign course of most patients with scoliosis of late onset has obscured the real efficacy of conservative treatment: a brace that obliterates the lumbar lordosis and prevents flexion ought to check progression if it was known that the curve would result in an unacceptable deformity.¹⁰

We need to know more about the natural history of scoliosis, however: we do not even know how many people with obvious deformity are performing quite satisfactorily in society.

Thus screening as currently performed uncovers vast numbers of inconsequential spinal asymmetries and a few patients with benign scoliosis of late onset. Moreover, it is ethically questionable to detect a cosmetic deformity of which the patient is usually unaware, to inform the patient that the problem stems from a spinal curvature of which the natural history is virtually unknown, and then to prescribe a "standard treatment" which is generally ineffective. On the rare occasions when screening in adolescence picks up a patient with untreated infantile progressive scoliosis the deformity may be so severe as to have caused irreversible organic damage already.

What then should be done? Firstly, we need to learn more about the epidemiological features of idiopathic scoliosis throughout life, and where screening is performed it should be directed to this end.^{37,40} It would not unduly tax existing arrangements, at least in Britain, to include an examination of the back during first immunisation at 3½ months and at the school medical examination at age 5 years to exclude the sinister scoliosis of early onset, for which prompt action is essential.⁴¹ Meanwhile, clinical research must be fostered in relation to the three dimensional nature of the deformity to produce more effective corrections for those who present later with a deformity.

Fortunately, there is one ray of real hope from apparent changes in natural history. The infantile progressive form has become much less common. When the condition was first described progressive curves were more prevalent than resolving ones.⁴² The next 30 years saw a dramatic reversal,⁴³ and now (possibly because babies are kept prone in their cots) the incidence of both varieties has considerably diminished.⁴⁴ A similar trend is seen in scoliosis of late onset, and where most screening has been performed the need for treatment has been less obvious, suggesting an even more benign course.⁴⁵

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¹ Goldberg C, Regan DF, Thompson F, Blake NS, Dowling F. Pilot study for a scoliosis screening project in south Dublin. *Ir Med J* 1980;73:265-8.

² Brooks HL, Azen SP, Gerberg E, et al. Scoliosis: a prospective epidemiological study. *J Bone Joint Surg* 1975;57A:968-72.

- ³ Lönstein JE. Screening for spinal deformities in Minnesota schools. *Clin Orthop* 1977;126:33-42.
- ⁴ Rogala EG, Drummond DS, Gurr J. Scoliosis: incidence and natural history. *J Bone Joint Surg* 1978;60A:173-6.
- ⁵ Torrell G, Nordwall A, Nachemson A. The changing pattern of scoliosis treatment due to effective screening. *J Bone Joint Surg* 1981;63A:337-41.
- ⁶ Adams W. *Lectures on the pathology and treatment of lateral and other forms of curvature of the spine*. London: Churchill & Sons, 1865.
- ⁷ Somerville EW. Rotational lordosis: the development of the single curve. *J Bone Joint Surg* 1952;34B:421-7.
- ⁸ Roaf R. The basic anatomy of scoliosis. *J Bone Joint Surg* 1966;48B:786-92.
- ⁹ Lawton JO, Butt WP, Dickson RA. Experimental idiopathic scoliosis. *J Bone Joint Surg* 1983;65B:657.
- ¹⁰ Dickson RA, Lawton JO, Archer IA, Butt WP. The pathogenesis of idiopathic scoliosis. *J Bone Joint Surg* 1984;66B:8-15.
- ¹¹ Dickson RA, Lawton JO, Archer IA, et al. Bi-planar spinal asymmetry: the pathogenesis of idiopathic scoliosis. *J Bone Joint Surg* 1984;66B:143-4.
- ¹² Bradford DS, Moe JH, Montalvo FJ, Winter RB. Scheuermann's kyphosis and round back deformity. Results of Milwaukee brace treatment. *J Bone Joint Surg* 1974;56A:740-58.
- ¹³ Carr WA, Moe JH, Winter RB, Lönstein JE. Treatment of idiopathic scoliosis in the Milwaukee brace. Long-term results. *J Bone Joint Surg* 1980;62A:599-612.
- ¹⁴ Bick EM, Copel JW, Spector S. Longitudinal growth of the human vertebra. A contribution to human osteology. *J Bone Joint Surg* 1950;32A:803-14.
- ¹⁵ Inkster RG. Osteology. In: Brash JC, ed. *Cunningham's textbook of anatomy*. 9th ed. London: Oxford Medical Publications, 1953:136.
- ¹⁶ Tupman ES. A study of bone growth in normal children and its relationship to skeletal maturity. *J Bone Joint Surg* 1962;44B:42-67.
- ¹⁷ Risser JC, Ferguson AB. Scoliosis: its prognosis. *J Bone Joint Surg* 1936;18:667-70.
- ¹⁸ James JIP. Idiopathic scoliosis: the prognosis, diagnosis and operative indications related to curve patterns and the age of onset. *J Bone Joint Surg* 1954;36B:36-49.
- ¹⁹ Hassan I, Björkrem I. Progression in idiopathic scoliosis after conservative treatment. *Acta Orthop Scand* 1983;54:88-90.
- ²⁰ Schultz AB, Hirsch C. Mechanical analysis of Harrington rod correction in idiopathic scoliosis. *J Bone Joint Surg* 1973;55A:983-92.
- ²¹ Thulbourne T, Gillespie R. The rib hump in idiopathic scoliosis. Measurement, analysis and response to treatment. *J Bone Joint Surg* 1976;58B:64-71.
- ²² Benson DR, DeWald RL, Schultz AB. Harrington rod distraction instrumentation. Its effect on vertebral rotation and thoracic compensation. *Clin Orthop* 1977;125:40-4.
- ²³ Aaro S, Dahlborn M. The effect of Harrington instrumentation on the longitudinal axis of rotation of the apical vertebra and on the spinal and rib-cage deformity in idiopathic scoliosis studied by computer tomography. *Spine* 1982;7:457-62.
- ²⁴ Dickson RA, Leatherman KD. Cotrel traction, exercises, casting in the treatment of idiopathic scoliosis. *Acta Orthop Scand* 1978;49:46-8.
- ²⁵ Edgar MA, Chapman RH, Glasgow MMS. Pre-operative correction in adolescent idiopathic scoliosis. *J Bone Joint Surg* 1982;64B:530-5.
- ²⁶ Leatherman KD, Dickson RA. Two-stage corrective surgery for congenital deformities of the spine. *J Bone Joint Surg* 1979;61B:324-8.
- ²⁷ Gonon GP, de Mauroy JC, Frankel P, Campo-Paysaa A, Stagnara P. Greffes antérieures en étai dans le traitement des cyphoses et cypho-scolioses. *Rev Chir Orthop* 1981;67:731-45.
- ²⁸ Zielke K, Berthel A. VDS-ventrale derotation spondylodese—vorläufiger bericht über 58 Fälle. *Beitr Orthop Traumatol* 1978;25:85-103.
- ²⁹ Luque ER. The anatomic basis and development of segmental spinal instrumentation. *Spine* 1982;7:256-9.
- ³⁰ Nachemson A. A long-term follow-up study of non-treated scoliosis. *Acta Orthop Scand* 1968;39:466-76.
- ³¹ Davies G, Reid L. Effect of scoliosis on growth of alveoli and pulmonary arteries and on right ventricle. *Arch Dis Child* 1971;46:623-32.
- ³² Kostuik JP, Israel J, Hall JE. Scoliosis surgery in adults. *Clin Orthop* 1973;93:225-34.
- ³³ Ponder CR, Dickson JH, Harrington PR, Erwin WD. Results of Harrington instrumentation and fusion in the adult idiopathic scoliosis patient. *J Bone Joint Surg* 1975;57A:797-801.
- ³⁴ Nilsson UK, Lundgren KD. Long-term prognosis in idiopathic scoliosis. *Acta Orthop Scand* 1968;39:456-65.
- ³⁵ Bengtsson G, Fallstrom K, Jansson B, Nachemson A. A psychological and psychiatric investigation of the adjustment of female scoliosis patients. *Acta Psychiatr Scand* 1974;50:50-9.
- ³⁶ Leaver JN, Alvik A, Warren MD. Prescriptive screening for adolescent idiopathic scoliosis: a review of the evidence. *Int J Epidemiol* 1982;11:101-11.
- ³⁷ Dickson RA. Scoliosis in the community. *Br Med J* 1983;286:615-8.
- ³⁸ Burwell RG, James NJ, Johnson F, Webb JK, Wilson YG. Standardised trunk asymmetry scores. *J Bone Joint Surg* 1983;65B:452-63.
- ³⁹ Dickson RA, Stamper P, Sharp AM, Harker P. School screening for scoliosis: cohort study of clinical course. *Br Med J* 1980;281:265-7.
- ⁴⁰ The British Orthopaedic Association and the British Scoliosis Society. School screening for scoliosis. *Br Med J* 1983;287:963-4.
- ⁴¹ Mehta MH, Morel G. The non-operative treatment of infantile idiopathic scoliosis. In: Zorab PA, Siegler D, eds. *Scoliosis 1979. Proceedings of the sixth symposium*. London: Academic Press, 1979:71-84.
- ⁴² Harrenstein RJ. Die Skoliose bei Säuglingen und ihre Behandlung. *Zeitschrift für Orthopädische Chirurgie* 1930;52:1-40.
- ⁴³ Lloyd-Roberts GC, Pilcher MF. Structural idiopathic scoliosis in infancy. *J Bone Joint Surg* 1965;47B:520-3.
- ⁴⁴ McMaster MJ. Infantile idiopathic scoliosis—can it be prevented? *J Bone Joint Surg* 1983;65B:612-7.
- ⁴⁵ Lönstein JE, Björklund S, Wanninger MF, Nelson RP. Voluntary school screening for scoliosis in Minnesota. *J Bone Joint Surg* 1982;64A:481-8.

STATURE AND IDIOPATHIC SCOLIOSIS. A PROSPECTIVE STUDY.

Archer IA, Dickson RA.

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Of the 5 303 children screened in the second epidemiological survey (Publication 3) 130 with spinal curvatures measuring 10° or more were studied prospectively with regard to height and growth velocity. Girls with adolescent idiopathic scoliosis measuring 15° or more were shown to be taller at each age than girls with smaller deformities and controls. Growth velocity was not dissimilar. Relative flattening of the back in the sagittal profile (see later under Pathogenesis) was put forward as one possible explanation for these differences in stature.

STATURE AND IDIOPATHIC SCOLIOSIS

A PROSPECTIVE STUDY

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A study of 130 scoliotic children with curves measuring 10° or more has been performed in order to elucidate the importance of stature, growth and development. Girls with adolescent idiopathic curves measuring 15° or more were taller than girls with smaller idiopathic curves and taller than those whose scoliosis was secondary to leg-length inequality (pelvic tilt scoliosis). No differences were observed as regards growth velocity or development.

The increased standing height may be genetic but the uncoiling effect of the normal kyphosis to give a flat lateral profile is a more likely cause. The familial trend in idiopathic scoliosis may therefore be explained by the genetically determined shape of the spine in the median (sagittal) plane.

The period of spinal growth is an important phase during which idiopathic scoliosis commences and can progress. Early reports of increased height in girls with adolescent idiopathic scoliosis (Willner 1974; Low *et al.* 1978) are of doubtful value, as the measurements were compared with those of historical controls, against which normal children today also would appear taller. Recent studies of adolescent English girls with idiopathic scoliosis (Dickson and Seviatt 1982) and of Southern Chinese girls (Leong *et al.* 1982) indicate that they are taller than contemporary controls, suggesting that an abnormal growth pattern might be responsible for the development and progression of the idiopathic deformity.

The idiopathic spinal deformity, a lateral curvature with rotation, is, however, secondary to a primary deformity in the median (sagittal) plane (Adams 1882; Somerville 1952; Dickson *et al.* 1983, 1984). In thoracic deformities the overall thoracic kyphosis is much reduced and at the apex of the curve there is a short-segment lordosis. This alteration in lateral profile has the effect of uncoiling the spine in the sagittal plane so that scoliotic patients appear taller even without any growth abnormality. To clarify the situation, stature, growth and development were studied in two groups of adolescent children—those with the radiographic deformity of idiopathic scoliosis and those without.

CHILDREN AND METHODS

Of 5303 children screened at school for the presence of a spinal deformity, 130 with lateral curvatures of the spine

measuring 10° or more agreed to take part in a longitudinal study of growth, maturity, and curve characteristics. Ninety-six children (75 girls and 21 boys) had adolescent idiopathic scoliosis, and 34 children (21 girls and 13 boys) had a non-structural lumbar curve, that is, a pelvic tilt scoliosis, secondary to leg-length inequality. The mean ages of the idiopathic and the pelvic tilt groups were 12.9 years (range 9.6 to 15.4) and 12.7 years (range 9.5 to 14.7) respectively.

All 130 children were assessed on three occasions—initially, six months later and one year later. On each occasion the curve characteristics—site, direction, and size (Whittle and Evans 1979)—were recorded from postero-anterior radiographs (low dosage techniques were used). Standing height was measured using a Harpenden stadiometer and the development of secondary sexual characteristics was rated by Tanner's method (1962). Bone age was determined from postero-anterior radiographs of the left hand and wrist by the method of Greulich and Pyle (1959)—this is referred to as the GP bone age—and by Tanner's second method (Tanner *et al.* 1975)—called the TW2 bone age.

Statistical significance was calculated using Student's unpaired *t*-test and the Mann-Whitney *U*-test.

RESULTS

Standing height. The mean standing height of girls with adolescent idiopathic scoliosis who had curves of 15° or more was significantly greater than that of the girls whose curves measured less than 15°, and also greater than that of the girls with pelvic tilt scoliosis; this difference occurred throughout the age range of 10 to 13 years ($P < 0.05$; Table I). The numbers of 9-year-old girls, of 15-year-old girls, and of boys, were too small to allow statistical analysis. There was no significant difference in the 14-year-olds, as two children with pelvic tilt scoliosis were exceptionally tall, one measuring 1.75 metres (on the 100th centile) and the other 1.70 metres (on the 95th

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Table I. Standing height and change in standing height of girls with adolescent idiopathic scoliosis (AIS) and girls with pelvic tilt scoliosis (PTS)

Age (years)	Mean standing height (metres)					Mean increase in standing height (metres)				
	Visit	All AIS	<15°	>15°	PTS	Visit	All AIS	<15°	>15°	PTS
10	1	1.43	1.42	1.48	1.46	1-2	0.027	0.024	0.037	0.046
	2	1.46	1.44	1.52	1.51	2-3	0.029	0.029	0.029	0.030
	3	1.49	1.47	1.55	1.54	1-3	0.056	0.053	0.066	0.077
11	1	1.51	1.47	1.57	1.44	1-2	0.029	0.029	0.029	0.070
	2	1.53	1.49	1.60	1.51	2-3	0.028	0.032	0.012	0.006
	3	1.56	1.52	1.66	1.54	1-3	0.053	0.055	0.048	0.017
12	1	1.53	1.53	1.55	1.49	1-2	0.054	0.060	0.036	0.035
	2	1.59	1.59	1.59	1.52	2-3	0.017	0.019	0.010	0.018
	3	1.60	1.61	1.60	1.54	1-3	0.071	0.079	0.046	0.053
13	1	1.59	1.58	1.61	1.57	1-2	0.021	0.023	0.014	0.045
	2	1.61	1.60	1.62	1.61	2-3	0.012	0.012	0.010	0.017
	3	1.62	1.62	1.63	1.62	1-3	0.033	0.035	0.025	0.059
14	1	1.60	1.60	1.61	1.64	1-2	0.013	0.014	0.012	0.018
	2	1.61	1.61	1.62	1.65	2-3	0.009	0.011	0.007	0.007
	3	1.62	1.62	1.63	1.66	1-3	0.023	0.024	0.019	0.025
15	1	1.66	1.63	1.60	No children	1-2	0.007	0.010	0.003	No children
	2	1.67	1.64	1.70	children	2-3	0.0	0.006	0.007	children
	3	1.67	1.64	1.74		1-3	0.007	0.004	0.011	

centile). When the standing heights of bone age-matched girls was analysed in relation to the site of the curve, again those with thoracic curves measuring 15° or more were found to be significantly taller than those whose thoracic curves were less than 15°, and also taller than those with pelvic tilt scoliosis; this was true on all three occasions when they were measured ($P < 0.05$). There were no significant differences in standing height with respect to curves at any other site ($P < 0.05$).

Table I shows the increase of standing height in the first and second six months (and by addition, the annual increase) in girls aged 10 to 15 years. There were no significant differences in growth velocity between girls with idiopathic scoliosis and those with pelvic tilt scoliosis ($P > 0.05$), nor between curves of different magnitudes ($P > 0.05$). When compared to the 1972 centile charts of Tanner and Whitehouse (1972) these girls appeared to be growing entirely normally and their growth charts were parallel to the centiles. Curve progression, measured by an increase in Cobb angle of 5° or more in the six months between measurements, occurred in 13 children with idiopathic scoliosis and this progressive trend was significant in girls with thoracic curves ($P < 0.05$). Curve regression, measured by a reduction in Cobb angle of 5° or more between measurements, occurred in 23 children with idiopathic scoliosis, but was not significant with regard to the sex of the child or to the site, size or direction of the curve ($P > 0.05$). Furthermore, no significant differences were observed in mean standing height or change in standing height between children with progressive, static or regressive curves ($P > 0.05$).

Bone age and puberty ratings. Figure 1 shows the mean initial GP bone age plotted against the mean initial chronological age for girls with idiopathic scoliosis,

demonstrating the very close correspondence between these observations. Figure 2 shows the mean initial TW2 bone age plotted against the mean initial chronological age for girls with idiopathic scoliosis; again there is very close correspondence, but compared with these less mature standards the girls naturally appear a little advanced at each age. There were no significant differences at any chronological age between the bone ages of children with idiopathic scoliosis and those with pelvic tilt scoliosis, neither in the girls nor in the boys at any of the three measurements ($P > 0.05$). No trends were observed for advancement of bone age to occur early in adolescence and retardation later.

No significant differences were observed at any time between the scores for the development of secondary sexual characteristics between children with idiopathic scoliosis and those with pelvic tilt scoliosis, nor between curves of different magnitudes, in either girls or boys ($P > 0.05$).

DISCUSSION

The period of the adolescent growth spurt is clearly important (Scammon 1927; Tanner 1962), particularly for those children with idiopathic scoliosis, because at that time progression seems most likely to occur (Bampfild 1824; Cobb 1948; Duthie 1959; Duval-Beaupère 1971). Growth studies in relation to adolescent idiopathic scoliosis have sought to blame an abnormal adolescent growth pattern in the production as well as the progression of the deformity (Willner 1974; Low *et al.* 1978; Leong *et al.* 1982). These studies, however, are of doubtful value, because they use historical controls for comparing both height and bone age; moreover, the method of calculating standing height also was questionable, in that it took no account of the three-dimensional

nature of the deformity. Because the secondary lateral curvature was the focus of attention, the standing height of children with adolescent idiopathic scoliosis was made even greater by applying a "correction factor" (Bjure, Grimby and Nachemson 1968), calculated as the increment of height that should be added if the lateral curvature was uncoiled. Paradoxically, it is uncoiling of the lateral profile of the spine which is associated with the production of the secondary scoliotic deformity and which accounts for the significant results of this present study, in which standing height measurements were real and not exaggerated. The increased height of girls with idiopathic scoliosis is entirely explicable on the basis of the flattened thoracic kyphosis; this flattening was originally observed by Adams (1882) and later confirmed by Willner (1981). The flatter the lateral profile the more is the rotational potential (Dickson *et al.* 1983, 1984). Thus girls with bigger thoracic curves are found to be taller than those with smaller curves. Incidentally, very small curves are so common as to be hardly abnormal (Burwell *et al.* 1982; Dickson 1983).

Despite the dubious observations concerning standing height of earlier reports, these were used to fuel the argument in favour of a primary growth abnormality (Willner 1974; Low *et al.* 1978; Leong *et al.* 1982). It was shown, however, that children with adolescent idiopathic scoliosis had no abnormalities referable to somatomedin or growth hormone status (Willner *et al.* 1976). The fact that in our study there were no significant differences in growth velocity or rate of maturation between children with idiopathic scoliosis and those with pelvic tilt scoliosis, confirms that there is no adolescent growth abnormality in children with adolescent

idiopathic scoliosis. The differences between TW2 bone age and the GP bone age merely reflect the different sources of the two atlases, GP bone age being derived from 1930s' upper-class Cleveland children who are still among the most advanced on record (Tanner 1962).

That standing posture rather than height is the critical factor also goes some way towards explaining the genetic background to idiopathic scoliosis. The familial trend observed in 1965 by Wynne-Davies (1968) has been confirmed by others (De George and Fisher 1967; Cowell, Hall and MacEwen 1972; Riseborough and Wynne-Davies 1973; Robin and Cohen 1975).

In 1951 Delmas studied lateral profiles and described three types of spinal morphology—subjects with marked, with intermediate or with slight curves in the median plane. Then Stagnara *et al.* (1982) measured normal lateral profiles and found enormous standard deviations around the mean for thoracic kyphosis and lumbar lordosis; he inferred that each individual has a genetically determined spinal physiognomy, just as he has a facial one. Willner (1981) demonstrated that children with idiopathic scoliosis have significantly flatter lateral profiles than normal children, and that these profiles in turn were flatter than those of patients with Scheuermann's disease. It is interesting that idiopathic scoliosis and Scheuermann's disease are opposite deformities in the sagittal plane, and have a not dissimilar community prevalence rate and familial trend (Sørensen 1964). The concept therefore arises that it may be the shape of the spine in the sagittal plane which is genetically determined, flat-backed children coming from flat-backed families and being vulnerable to idiopathic scoliosis, and round-backed children coming

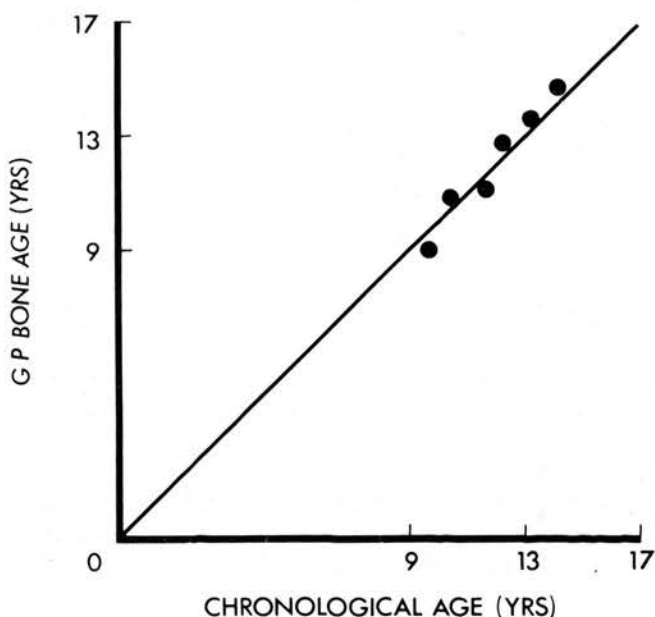


Fig. 1

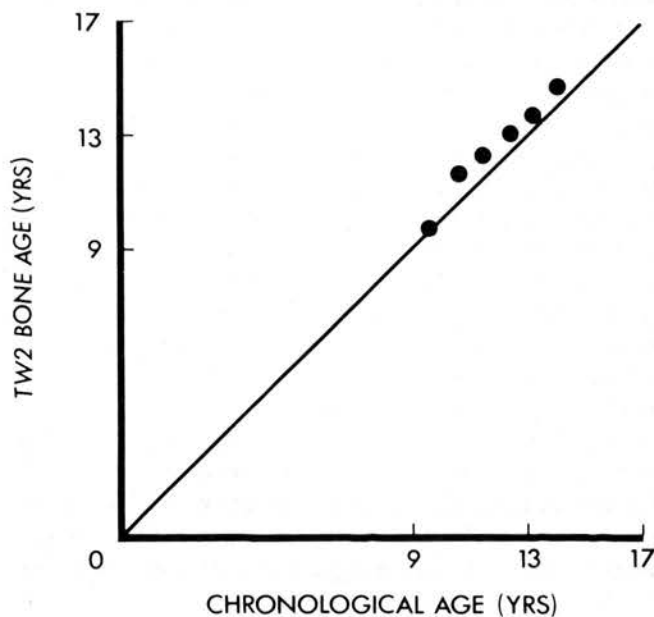


Fig. 2

Mean initial bone age plotted against mean initial chronological age for girls with adolescent idiopathic scoliosis. Figure 1—GP bone age determined by the method of Greulich and Pyle (1959). Figure 2—TW2 bone age determined by the second method of Tanner *et al.* (1975).

from round-backed families being vulnerable to the development of Scheuermann's disease. This is supported by the very close correspondence between family members as regards growth and shape, rising through the degrees of relationship to the highest values in identical twins (Bolk 1923; Boas 1932; Sontag and Nelson 1933; Hewitt 1957; Tanner 1962); this correspondence must also include spinal shape in the sagittal plane. Thus the inherited factor accounting for the familial trend in adolescent idiopathic scoliosis, and previously unexplained, may be nothing more than the flattened lateral profile.

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REFERENCES

- Adams W. *Lectures on the pathology and treatment of lateral and other forms of curvature of the spine*. 2nd ed. London: Churchill & Sons, 1882.
- Bampfild RW. *An essay on curvature and diseases of the spine, including all the forms of spinal distortion*. London: Longman, Hurst, Rees, Orme, Brown & Green, 1824. Cited by Risser JC. Scoliosis: past and present. *J Bone Joint Surg [Am]* 1964;46-A:167-99.
- Bjure J, Grimby C, Nachemson A. Correction of body height in predicting spirometric values in scoliotic patients. *Scand J Clin Lab Invest* 1968;21:191-2.
- Boas F. Studies in growth. *Hum Biol* 1932;4:307-50.
- Bolk L. The menarche in Dutch women and its precipitated appearance in the youngest generation. *Proc Acad Sci Amst Sec Soc* 1923;26:650-63.
- Burwell RG, James NJ, Johnson F, Webb JK. The rib hump score: a guide to referral and prognosis? *J Bone Joint Surg [Br]* 1982;64-B:248.
- Cobb JR. Outline for the study of scoliosis. *Am Acad Orthop Surg Instr Course Lect* 1948;5:261-75.
- Cowell HR, Hall JN, MacEwen GD. Genetic aspects of idiopathic scoliosis: a Nicholas Andry Award essay. *Clin Orthop* 1972;86:121-31.
- De George FV, Fisher RL. Idiopathic scoliosis: genetic and environmental aspects. *J Med Genet* 1967;4:251-7.
- Delmas A. Types rachidiens de statique corporelle. *Rev Morphophysiol hum* 1951;4(11):26-32.
- Dickson RA. Scoliosis in the community. *Br Med J* 1983;286:615-8.
- Dickson RA, Lawton JO, Archer IA, Butt WP. The pathogenesis of idiopathic scoliosis: biplanar spinal asymmetry. *J Bone Joint Surg [Br]* 1984;66-B:8-15.
- Dickson RA, Lawton JO, Archer IA, et al. Combined median and coronal plane asymmetry: the essential lesion of progressive idiopathic scoliosis. *J Bone Joint Surg [Br]* 1983;65-B:368.
- Dickson RA, Sevitt EA. Growth and idiopathic scoliosis: a longitudinal cohort study. *J Bone Joint Surg [Br]* 1982;64-B:385.
- Duthie RB. The significance of growth in orthopaedic surgery. *Clin Orthop* 1959;14:7-19.
- Duval-Beaupère G. Pathogenic relationship between scoliosis and growth. In: Zorab PA, ed. *Scoliosis and growth: proceedings of a third symposium*. Edinburgh and London: Churchill Livingstone, 1971:58-64.
- Greulich WW, Pyle SI. *Radiographic atlas of skeletal development of the hand and wrist*, 2nd ed. Stanford: Stanford University Press; and London: Oxford University Press, 1959.
- Hewitt D. Some familial correlations in height, weight and skeletal maturity. *Ann Hum Genet* 1957;22:26-35.
- Leong JCY, Low WD, Mok CK, Kung LS, Yau ACMS. Linear growth in Southern Chinese female patients with adolescent idiopathic scoliosis. *Spine* 1982;7(5):471-5.
- Low WD, Mok CK, Leong JCY, Yau ACMS, Lisowski FP. The development of Southern Chinese girls with adolescent idiopathic scoliosis. *Spine* 1978;3(2):152-6.
- Riseborough EJ, Wynne-Davies R. A genetic survey of idiopathic scoliosis in Boston, Massachusetts. *J Bone Joint Surg [Am]* 1973;55-A:974-82.
- Robin GC, Cohen T. Familial scoliosis: a clinical report. *J Bone Joint Surg [Br]* 1975;57-B:146-7.
- Scammon RE. The first serial study of human growth. *Am J Phys Anthropol* 1927;10:329-36.
- Somerville EW. Rotational lordosis: the development of the single curve. *J Bone Joint Surg [Br]* 1952;34-B:421-7.
- Sontag LW, Nelson VL. A study of identical triplets: comparison of the physical and mental traits of a set of monozygotic dichorionic triplets. *J Hered* 1933;24:473-80.
- Sørensen HL. *Scheuermann's juvenile kyphosis: clinical appearances, radiography, aetiology and prognosis*. Copenhagen: Munksgaard, 1964.
- Stagnara P, De Mauroy JC, Dran G, et al. Reciprocal angulation of vertebral bodies in a sagittal plane: approach to references for the evaluation of kyphosis and lordosis. *Spine* 1982;7(4):335-42.
- Staub HA. Eine Skoliotikerfamilie. Ein Beitrag zur Frage der kongenitalen Skoliose und der Heredität der Skoliosen. *Z Orthop Chir* 1922;43:1-20.
- Tanner JM. *Growth at adolescence: with a general consideration of the effects of hereditary and environmental factors upon growth and maturation from birth to maturity*. 2nd ed. Oxford: Blackwell Scientific, 1962.
- Tanner JM, Whitehouse RH. *Growth and development chart*. Hertford: Castlemead Publications, 1972.
- Tanner JM, Whitehouse RH, Marshall WA, Healy MJR, Goldstein H. *Assessment of skeletal maturity and prediction of adult height (TW2 method)*. London: Academic Press, 1975.
- Whittle MW, Evans M. Instrument for measuring the Cobb angle in scoliosis. *Lancet* 1979;i:414.
- Willner S. A study of growth in girls with adolescent idiopathic structural scoliosis. *Clin Orthop* 1974;101:129-35.
- Willner S. Spinal pantograph: a non-invasive technique for describing kyphosis and lordosis in the thoraco-lumbar spine. *Acta Orthop Scand* 1981;52:525-9.
- Willner S, Nilsson KO, Kastrup K, Bergstrand CG. Growth hormone and somatomedin A in girls with adolescent idiopathic scoliosis. *Acta Paed Scand* 1976;65(5):547-52.
- Wynne-Davies R. Familial (idiopathic) scoliosis: a family survey. *J Bone Joint Surg [Br]* 1968;50-B:24-30.

SCOLIOSIS IN THE COMMUNITY.

Dickson RA, Archer IA.

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London etc, 77-100, 1984.

This textbook chapter critically analysed scoliosis in the community with particular reference to definitions and criteria, untreated scoliosis, treatment, and the spectrum of scoliosis in the community.

3

Scoliosis in the community

Robert A. Dickson and I. A. Archer

INTRODUCTION

'Screening' for idiopathic scoliosis was first performed in the early 1960s in Delaware (Cronis and Russell, 1965) although surveys of large numbers of chest films taken by mass miniature radiography for tuberculosis had, however, been carried out at least 20 years earlier, providing information concerning the prevalence rate of thoracic scoliosis (Shands and Eisenberg, 1955). Since then screening for scoliosis has mushroomed and gained worldwide enthusiasm. *Table 3.1* summarizes some of the recent data derived from school screening (Kane and Moe, 1970; Strayer, 1973; Sells and May, 1974; Segil, 1974; Golomb and Taylor, 1975; Brooks *et al.*, 1975; Span, Robin and Makin, 1976; Inoue, 1977; Abbott, 1977; Flynn, Riddick and Keller, 1977; Newman and DeWald, 1977; Bellyei *et al.*, 1977; Ascani, Salsano and Giglio, 1977; O'Brien and van Akkerveken, 1977; Adair, Van Wijk and Armstrong, 1977; Lonstein, 1977; Rogala, Drummond and Gurr, 1978; Smyrnis *et al.*, 1979; Dickson *et al.*, 1980). Unfortunately most of these reports do not make the best use of the data obtained. The gaps in *Table 3.1* indicate where there is insufficient information provided even to determine the prevalence rate of scoliosis at simply defined curve magnitudes.

Nonetheless, reading between the lines, a trend is apparent that a substantial proportion of samples screened, of the order of 15%, do show evidence of a trunk asymmetry on crude visual testing and that this proportion decreases exponentially with increasing curve magnitude until only approximately two to five individuals per 1000 screened have curves measuring 20° or more, for whom treatment is customarily considered.

The situation, however, is far from being straightforward, and the most controversial areas are definitions and criteria for screening, the consequences of untreated idiopathic scoliosis, the treatment of idiopathic scoliosis, and the natural history of scoliosis in the community.

DEFINITIONS AND CRITERIA

There is no doubt that screening for scoliosis in schools or anywhere else is designed to detect early the condition of adolescent idiopathic scoliosis. Idiopathic scoliosis is a lateral curvature of the spine with rotation in the absence of any associated congenital abnormality or musculoskeletal condition. While rotation is very much more obvious with idiopathic scoliosis, it can be shown quite clearly

Table 3.1 Scoliosis prevalence rates from screening

Author	Year	Positive %	Cobb angle degrees (%)			
			5	10	15	20
Kane and Moe	1970	—	—	—	—	0.13
Strayer	1973	—	—	5.0	—	2.0
Sells and May	1974	1.6	—	—	—	—
Segil	1974	—	—	2.5	—	—
Golomb and Taylor	1975	8.5	—	—	—	—
Brooks <i>et al.</i>	1975	—	13.6	—	—	0.2
Span, Robin and Makin	1976	13.0	—	1.5	—	—
Inoue	1978	13.2	—	3.8	—	0.7
Abbott	1977	3.4	—	—	—	0.13
Flynn, Riddick and Keller	1977	1.8	—	—	—	—
Ascani, Salsano and Giglio	1977	4.1	3.4	—	0.4	0.2
O'Brien and Van Akkerveken	1977	15.0	—	2.0	—	—
Adair, Van Wijk and Armstrong	1977	20.0	11.9	—	—	—
Lonstein	1977	8.3	—	4.0	—	—
Rogala, Drummond and Gurr	1978	4.5	2.37	1.88	—	0.29
Smyrnis <i>et al.</i>	1979	10.0	2.31	1.63	0.63	0.43
Dickson <i>et al.</i>	1980	8.3	—	2.5	—	0.25

biomechanically that any lateral curvature of the spine is associated with rotation, albeit of small magnitude (White, 1971) (*Figure 3.1*). As will be seen, half of all scolioses detected in the community are not idiopathic at all and the presence of other infinitely less important scolioses dilutes the intensity and confuses the interpretation of epidemiological surveys (Dickson *et al.*, 1980; Dickson, 1983). Furthermore, for some inexplicable reason, idiopathic scoliosis has been divided into three groups according to age of onset, presumably due to three phases of increased growth velocity, only one of which in fact exists (Tanner, 1962). *Figure 3.2* shows the velocity of growth with age, and it can be seen that the only time a child increases its growth velocity from the time of 4 months *in utero* is during adolescence. However, it is not the basis for this arbitrary division which is important but the fact that the division itself is not strictly adhered to. A child of 11 with a 60° curve does not have adolescent idiopathic scoliosis as the condition most certainly started well before the age of 10, and this point is crucial not only when considering the age of selection for screening but also as regards the consequences of untreated scoliosis.

Screening is defined as the presumptive identification of unrecognized disease or defect by the application of tests, examinations or other procedures which can be applied rapidly (Commission on Chronic Illness, 1957). A prerequisite for screening is that the natural history of the condition under scrutiny should be adequately understood (Whitby, 1974). This is manifestly not the case with idiopathic scoliosis, about which little is known. Screening tests sort out apparently well persons who have a disease from those who probably do not, and screening tests are not intended to be diagnostic. Persons with positive or suspicious findings must be

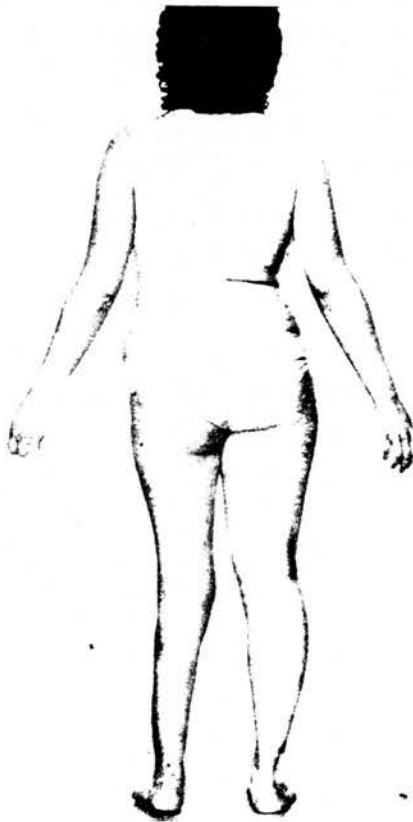


Figure 3.1 Pelvic tilt scoliosis due to a leg length inequality from poliomyelitis: there is a lumbar curve convex to the left and even in the erect position rotation is obvious with the left flank more prominent posteriorly

referred to their physicians for diagnosis and treatment. The screening programmes listed in *Table 3.1* would have been better described as epidemiological surveys (which make measurements on defined populations in order to elucidate prevalence rate, incidence rate and natural history of the variable under study).

When screening is carried out on selected subgroups of the population (selected as being at relatively high risk on the basis of epidemiological research) it is called 'selective screening'. The reports listed in *Table 3.1* could be described as epidemiological surveys (of a sort) derived by selective screening. Screening for

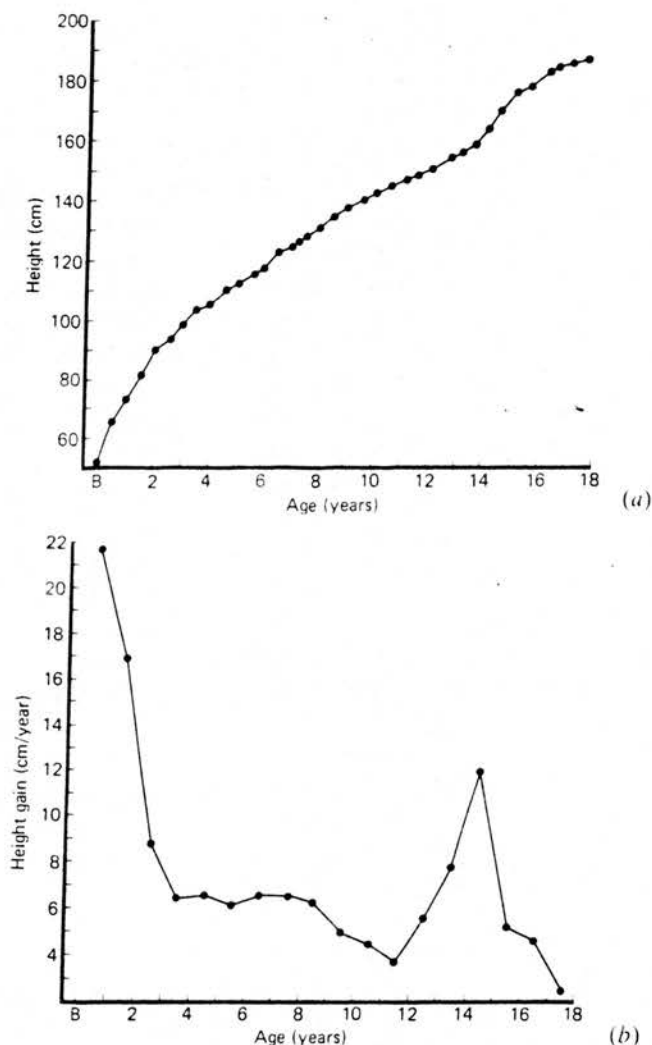


Figure 3.2 (a) Plotting standing heights from year to year produces a flat graph suppressing the speed of growth. (b) Plotting height gain from year to year produces the much more meaningful growth velocity graph

scoliosis selects by age, the 10–14-year-olds being considered particularly vulnerable and therefore providing an adequate harvest, but of what? (Figure 3.3). That this particular age group is at relatively high risk is merely conjectured on the basis of the so-called adolescent growth spurt. The selection process should be soundly based on epidemiological research and later when natural history is discussed it will be seen that there is little scientific evidence supporting the concept that the 10–14 year age group should necessarily be the optimal age group to screen.

An irritating aspect of the majority of screening reports concerns the misuse of words, 'incidence' and 'prevalence' in particular, which are frequently used as

freely interchangeable words of the same meaning. This is of course is not the case: the proper terms are 'incidence rate' and 'prevalence rate' (Petrie, 1978). Concerning scoliosis the incidence rate is the number of individuals developing a scoliosis in a specified period of time as a proportion of the total number exposed to that risk, while the prevalence rate refers to the number of individuals with a scoliosis existing at any time as a proportion of the number exposed to that risk. Therefore the data in *Table 3.1* concern prevalence rates and, with a condition that may progress, but usually remains static or regresses, the only manner in which we shall

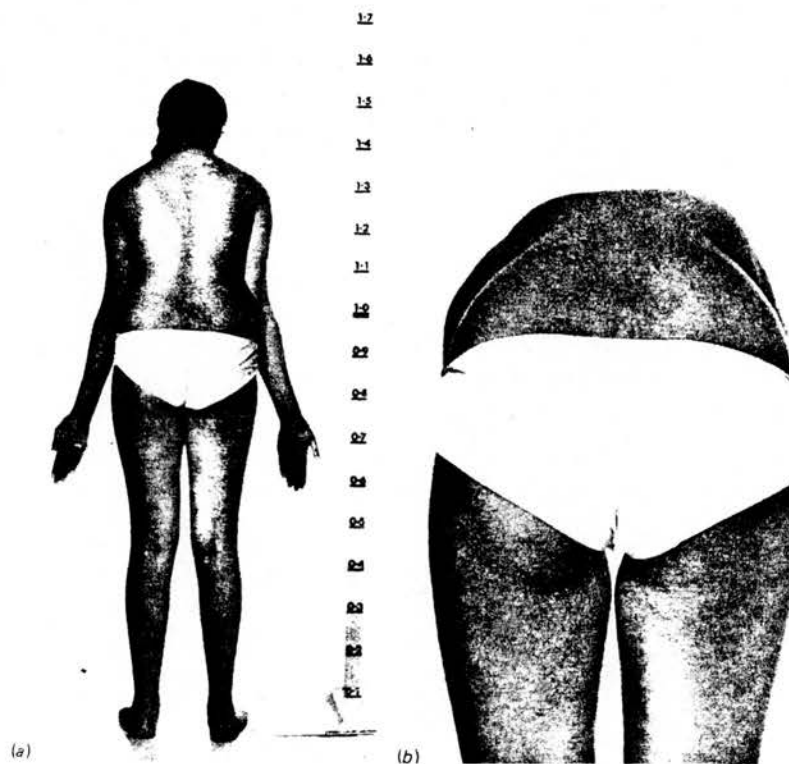


Figure 3.3 (a) In the erect position the spine is perfectly straight. (b) On forward bending there is a normal minimal right rib hump due to the asymmetry of the thoracic prisms (see Chapter 1 for mechanical explanation)

learn anything to do with incidence rate is by taking a sample population of, say, 9-year-olds and following them through their subsequent growth, noting the annual proportion of fresh cases.

When screening expands in the way it has done over the past 10 years it would be expected that its validity (the ability of the test to separate those who have the disease from those who do not), the sensitivity (the ability of the test to give a positive finding when the person truly has the disease under study), and specificity (the ability of the test to give a negative finding when the person tested is free of the

disease under study) (Whitby, 1974) would be accurately documented. This will certainly be necessary before contemplating mass screening on a national scale. It must also be said that while the method of screening remains essentially the same, a crude visual inspection of the child's back in different postures, the degree of training of the screener has varied considerably, which must affect validity, sensitivity and specificity of the procedure (Leaver, Alvik and Warren, 1982). That the screening procedure is by crude visual examination may be the most important aspect of screening as with a more accurate method substantially more and probably all growing children would show evidence of coronal plane asymmetry of the spine (Sabatier, 1777) in which case, analogous to screening for breast cancer, we are screening for the presence of the breasts and not for the underlying pathological breast mass.

UNTREATED SCOLIOSIS

A crucial part of the argument used by those in favour of screening is the effect of untreated scoliosis which appears to so adversely affect such individuals. *Table 3.2* lists these consequences with their proponents (Nachemson, 1968; Nilsonne and

Table 3.2 Consequences of untreated scoliosis

<i>Consequences</i>	<i>Author</i>	<i>Year</i>
Mortality	} Nachemson Nilsonne and Lundgren Collis and Ponseti	1968
Morbidity		1968
		1969
Social factors	Nilsonne and Lundgren Bengtsson <i>et al.</i>	1968 1974
Disability compensations	} Dahlberg and Nachemson	1977
Pensions		
Employment		
Economic loss		
Pain	Ponseti and Friedman	1950
	Nilsonne and Lundgren	1968
	Nachemson	1968
	Nachemson (personal communication)	1980

Lundgren, 1968; Collis and Ponseti, 1969; Bengtsson *et al.*, 1974; Dahlberg and Nachemson, 1977). Superficially, it makes compulsive viewing but there is probably much less of relevance to screening. While these mainly Swedish studies did their best to quantify the effects of untreated scoliosis, serious doubts would have to be raised concerning whether their inferences concern adolescent idiopathic scoliosis, which is the condition screened for when the selection process focuses on

10–14-year-olds. Furthermore, these are essentially historical studies and it would be anachronistic to think that the magnitude of these problems in the western world at present is anything like what it was half a century ago in terms of both primary and secondary medical care.

Since Hippocrates clearly described the respiratory handicap of severely scoliotic patients, their pulmonary problems have been frequently reported. Such problems are notorious with progressive congenital scolioses, some neuromuscular scolioses and the progressive idiopathic cases of particularly early onset, either infantile or early juvenile (*Figure 3.4*). There is, however, no convincing evidence that adolescent idiopathic scoliosis, by far the most common spinal deformity and the one specifically screened for, is associated with any reduction in respiratory



Figure 3.4 Anteroposterior radiograph of a severe idiopathic deformity of early onset; this is the age of onset associated with organic health problems

function. Several long-term studies of patients with adolescent-onset idiopathic scoliosis clearly demonstrate that pulmonary function remains essentially normal even if curve magnitude is in excess of 100° (Kostuik, Israel and Hall, 1973; Ponder *et al.*, 1975; Dickson and Leatherman, 1976). To date Lynn Reid has provided the only acceptable explanation for this in that the pulmonary parenchyma is fully developed by the age of 8 years and thereafter only grows (Reid, 1971). If a significant chest deformity occurs before that period then pulmonary function may certainly be in jeopardy whereas a comparable size deformity developing after this time has much less effect. In this respect the 60° often quoted as a threshold for the appearance of pulmonary problems (Collis and Ponseti, 1969) is of relevance only when the age of onset of this size deformity is also considered.

For these reasons the most quoted studies (Nachemson, 1968; Nilsonne and Lundgren, 1968) concerning mortality and morbidity must be particularly strictly interpreted. Although the 102 untreated scoliotic patients traced by Nilsonne and Lundgren appear to have been idiopathic, curve magnitude was greatly in excess of that associated with adolescent onset idiopathic scoliosis, indicating that a significant number of patients must have had their curve onset in infancy or the early juvenile period. It is therefore not surprising that they found a mortality rate twice as high as for the general population, with cardiac or pulmonary disease accounting for 60% of these deaths. The real importance of these studies is therefore often overlooked and this is the problem of selecting the appropriate age for screening. While the 10–14 year age group provides large numbers of small curves, the condition is much more benign than that occurring earlier. If selection for screening included, say, 7, 8 and 9-year-olds, then the prevalence rate for scoliosis would be smaller at these ages in the authors' opinion, but the prevalence rate of really important cases would be relatively greater.

Nachemson's study of 130 untreated scoliotic patients provided similar data concerning mortality rate and morbidity but did include a substantial number of congenital and other obviously non-idiopathic cases, thus diluting the intensity of the data even further. However, 52 patients who were considered to have adolescent idiopathic scoliosis fared much better than the rest, with a cardiopulmonary death rate not dissimilar from that which would be expected in a control population. This strongly supports the benignity of the adolescent onset case. Collis and Ponseti (1969) attempted to study 358 patients with idiopathic scoliosis but were only able to examine 30% of them, which markedly diminishes the importance of the inferences in a retrospective study. Their claim that two-thirds of patients with thoracic curves greater than 60° had diminished vital capacity was taken up by many as a reason for surgically treating idiopathic scoliosis at this threshold. The authors feel that the correct interpretation of these data is that the majority of patients had early juvenile onset idiopathic scoliosis with curves of significant magnitude even before the age of 8 years.

Similarly, data concerning disability compensations, pensions and employment are not strictly relevant to adolescent idiopathic scoliosis although both Swedish studies noted a decreased ability to perform ordinary work. As seems likely, both the pulmonary and economic problems arise in the same individuals with early onset curves and not with regard to adolescent idiopathic scoliosis, which further removes their relevance from screening programmes.

Perhaps the most important and relevant data from these studies concern social factors (Bengtsson *et al.*, 1974; Nilsonne and Lundgren, 1968). A high proportion of untreated scoliotics do not marry, and those that do have a higher than normal divorce rate, with fewer children per marriage and increased psychiatric consultation and suicide rates. Although this again strictly pertains to those severe curves of earlier onset, it does attempt to quantify the social consequences of significant asymmetry of body topography (*Figure 3.5*). Certainly in the absence of any evidence that adolescent onset idiopathic scoliosis is associated with pulmonary or economic insufficiency, the one clear problem that these children have is body disfigurement and even if the magnitude of these social variables is not so great as

with juvenile onset idiopathic scoliosis, it is nonetheless a problem shared by all patients and is the only complaint with which they present.

Some reports indicate that the prevalence rate and severity of back pain are increased with a scoliotic spine and others suggest that there is no difference from straightbacked counterparts (Collis and Ponseti, 1969; Nachemson, 1968; Nilssonne and Lundgren, 1968). Whether or not pain is more frequent or more severe, it is certainly more difficult to eradicate from the scoliotic spine when it does appear and traditional conservative measures frequently either fail or require to be repeated on many occasions before relief is obtained (Dickson and Leatherman,

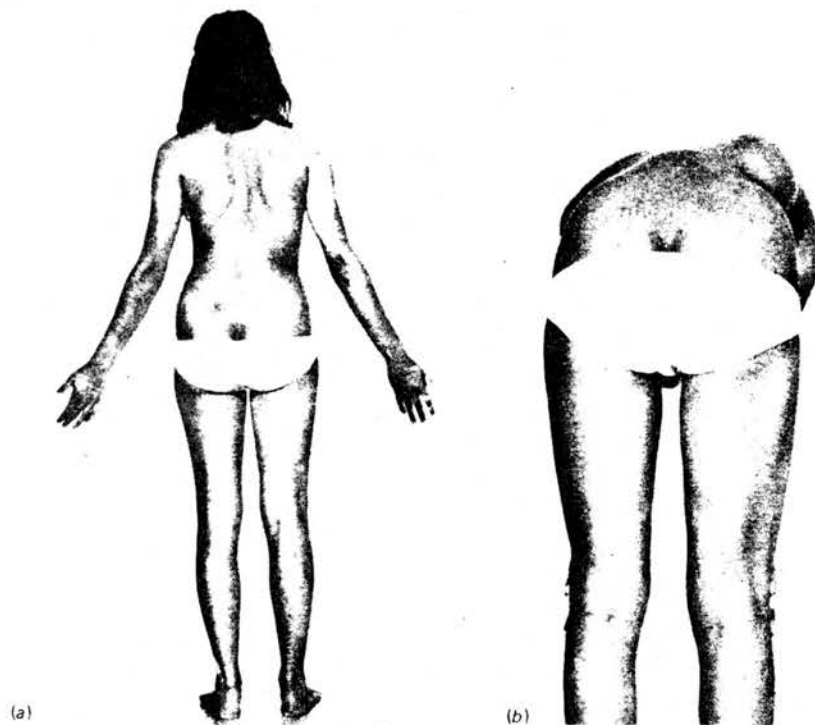


Figure 3.5 Late onset idiopathic scoliosis: (a) erect; (b) bending forward – a problem only of asymmetry of body topography with possible psychological and social implications

1976). In this respect posterior fusion with Harrington instrumentation is recommended by many as the definitive treatment for a painful scoliotic spine in the adult (Dickson and Leatherman, 1976; Kostuik, Israel and Hall, 1973; Ponder *et al.*, 1975).

Another strong argument used in favour of screening programmes is the economic loss to the community as a consequence of untreated scoliosis, and this is probably true. Dahlberg's econometric analysis determined that if a child of 10 years with a 30° curve who should be braced does not get the benefit of treatment then it will ultimately cost the community a factor of 90. While this is an apparently staggering statistic, the real importance of this observation is that even a child with

a curve as small as 30° at the age of 10 does not have adolescent idiopathic scoliosis, as the curve certainly started during the juvenile period. From the foregoing it is clear that scoliosis of early onset is a troublesome condition and it is also obvious that screening adolescents will uncover curves that originate in the juvenile period. This implies, however, that the uncovering process is almost certainly too late. It also implies a significant number of cases of untreated juvenile onset scoliosis in the western world, and therefore a careful study of untreated scoliosis at the present time in relation to mortality, morbidity, social and economic factors is clearly essential before screening can be recommended on a national scale.

TREATMENT OF IDIOPATHIC SCOLIOSIS

Despite uncertain knowledge of the natural history of idiopathic scoliosis, 'standard' treatment methods have evolved according to curve size with relatively predictable results. *Table 3.3* summarizes the treatment of idiopathic scoliosis which is practised by the majority of scoliosis surgeons. Of course, individual surgeons alter the thresholds to suit their own interpretation of natural history which is just as arbitrary as the figures in *Table 3.3*. It is well established that

Table 3.3 Treatment of idiopathic scoliosis

<i>Cobb angle</i>		<i>Treatment</i>
0-20		Nil
20-45	Thoracic	Brace
20-60	Lumbar or double structural	Brace
45+	Thoracic	Surgery
60+	Lumbar or double structural	Surgery

rationalizing treatment of a three-dimensional deformity based upon a simple measurement on a two-dimensional radiograph is open to question. Nonetheless, with a sufficient pool of patients curve magnitude does reflect the deformity, but as regards the individual case this is certainly not so. Few would dispute the need to instrument and fuse an idiopathic curve in the thoracic region of 60° although equally few would be happy about obtaining at best a 50% correction of curve magnitude and a substantially smaller correction of the rotational prominence with which the child presented. There are, however, many individuals with curves of only 35° with a significant rotational deformity, in which case, unless the surgeon ignores the thresholds and recommends operation on his own visual judgement, he will either leave it if the patient is beyond peak adolescent growth velocity or wait until it gets to the required magnitude so that treatment can be relatively less effective.

It is traditional not to treat curves measuring less than 20° . Quite why this particular figure has been selected is unclear, although it does represent 'a nice

round figure'. If a curve measuring 14° was destined to finish untreated at 90° it would obviously be inappropriate to give it 6° start before applying treatment. Clearly much more needs to be known concerning the natural history of idiopathic scoliosis with particular reference to factors indicating a poor prognosis (*see below*).

Bracing is the mainstay of conservative treatment and is recommended for curves between 20° and 45° . There is no evidence that either non-skeletal traction (Dickson and Leatherman, 1978) or electrosplinal stimulation in any way affect natural history. Unfortunately, bracing is in no way corrective and long-term studies indicate that an optimal result from brace-wearing is when the curve measures exactly the same at maturity plus 1 year as it did when treatment started (Carr *et al.*, 1980). Even more alarming is the observation that the curve is odds on to progress until the mid-20s (Hassan and Bjerkreim, 1983), although this is less surprising on consideration that the vertebral epiphyses do not fuse until the 25th year anyway (Inkster, 1953). Furthermore, this ability to prevent progression only exists with relative certainty for curves between 20° and 30° and thereafter up to 45° curve deterioration is progressively less well prevented. Indeed, because follow-up studies of curves measuring between 30° and 45° do not include important data concerning biological growth (an up-to-date skeletal age measure such as the Tanner and Whitehouse second method, standing height, sitting height, weight, puberty ratings, etc.), there is no reliable evidence that brace-wearing alters the natural history of these curves. The only biological growth data contained in these studies was the Gruelich and Pyle bone age (Gruelich and Pyle, 1959) which was derived from upper-class 1930s Cleveland children, who were among the most advanced children on record. By comparison with these standards a 'normal' child would appear to be 2 years younger than he or she really is.

More often used are status of ossification and fusion of the iliac crest apophysis (Risser's sign) (Risser, 1958), and the status of ossification of the vertebral ring epiphyses. The former is only measurably discernible within a few years of maturity of the pelvis, which occurs at 16 years in girls and 18 years in boys and has no relevance to spinal maturity. The vertebral epiphyses become indiscernible well before the end of spinal growth (Inkster, 1953). Even if these were useful determinants of spinal maturity, they are of no value in determining biological age during the much more important phase of earlier growth.

Brace-wearing as a truly preventive measure is therefore primarily directed to individuals whose curves are between 20° and 30° . This is where current treatment practices are extremely important in relation to school screening for scoliosis, which throws up large numbers of small magnitude curves of which only a very few have any progression potential. Recent careful longitudinal cohort studies demonstrated that less than one-fifth of curves between 20° and 30° progressed (increased in curve magnitude by 5° or more) during the course of 1 year (Dickson *et al.*, 1980; Dickson, 1983). Yet one of the major problems resulting from screening is the great increase in the number of braces, and for that matter brace-makers, required to boost an already maximally overstretched establishment (at least as far as the UK is concerned, with a health service dependent upon a finite sum of money) in order to cope with these small curves. While it can be argued ethically that a brace should not be withheld from a child whose curve progresses beyond 20° , it is also clear that

dispensing braces for curves of that magnitude treats the whole for the benefit of less than 20%. In addition, such a practice greatly enhances the figures for the efficacy of brace-wearing when more than four-fifths of the excellent results occur in patients who would not have progressed significantly if untreated. Furthermore, it is customary before prescribing a brace to have Cobb angle evidence of curve progression of at least 5°, which narrows the band of safety to 25°–30°. Also a curve demonstrating a 5° progression before treatment may have exhausted all its significant progression potential, thus reducing the figure for progression of one-fifth to an even smaller fraction. Much more epidemiological research is clearly indicated before the above confusing data concerning treatment can be properly rationalized, and this provides strong evidence against the use of screening for anything else than to provide material for these very necessary natural history studies, including a controlled clinical trial of brace treatment.

Despite all the uncertainties concerning the true magnitude of untreated scoliosis and the methods of treatment, there is one really disquieting statistic, and that is that the average curve magnitude at presentation is of the order of 50° in the UK (Belstead and Edgar, 1978). This immediately puts a significant number of children straight into the surgical treatment category and the majority into the brace category with curves bigger than can be expected to be controlled adequately by such treatment. Although screening for breast cancer has only been performed on small population samples, it has led to increased public knowledge with many more women becoming aware of the importance of self-examination and therefore many more presentations with earlier stage disease. Similarly with idiopathic scoliosis, where screening has been performed, even on a very limited basis, average curve magnitude at presentation has dropped to the more acceptable figure of about 35° (Torell, Nordwall and Nachemson, 1981) and cannot be attributed directly to screening as only very small numbers of the population at risk have been examined. This supposed spin-off of increased community awareness is a very important factor and one that should be fostered nationally. Indeed, this approach would be much more desirable than escalating screening and would not overburden an already maximally stretched establishment with a welter of manifestly non-idiopathic cases. Unfortunately the reduction of average curve magnitude is almost certainly not attributable to increased community awareness, but is due to a sharp increase in small magnitude curves derived by screening. This makes the frequency distribution bimodal, thus reducing the mean but having no effect on the 50° mode.

Low-dose radiographic technique

With the vast increase that is occurring in screening worldwide it is vital that radiation dosage to growing children should be acceptably low. It is obviously an extremely important ethical question when the great majority of children X-rayed as a result of screening do not have progressive idiopathic scoliosis. Only a few groups have, however, paid strict attention to this matter (Ardran *et al.*, 1980; DeSmet, Fritz and Asher, 1981; Gray, Hoffman and Peterson, 1983), and the

Oxford Scoliosis Study Group has evolved the lowest dose technique. Furthermore, the matter was discussed with expert radiation physicists before screening was started. When an anteroposterior X-ray of the spine is taken under traditional conditions there is an unacceptably high radiation dosage received by the developing breast and thyroid, the two most important organs at this stage in development that cannot be shielded (*Table 3.4*). By conventional techniques the spine is X-rayed from the front with a focus-film distance of 1.2 m (4 ft). By the use of a fast

Table 3.4 Mean radiation dosages by conventional and low dose techniques (Ardran *et al.*, 1980)

<i>Routine anteroposterior projection</i>	
Standard screens	
Sternal skin	230 mrad
Quanta II screens	
Sternal skin	96 mrad
<i>Low dose technique</i>	
Sternal skin	19 mrad
Breast	1.6 mrad
Thyroid	1.2 mrad

film-screen combination (Quanta II screens) the dosage is reduced by more than half. Then, if the X-ray is taken from the back, the whole of the anteroposterior width of the torso absorbs a significant quantity of the dose before it reaches the breast and thyroid. Finally, if the focus-film distance is increased to 3.6 m (12 ft) with the incorporation of an antiscatter grid, the final minimum radiation dosage is achieved (*Figure 3.6*). This is less than 2% of that obtained with conventional techniques and is similar to the annual background radiation dosage in many parts

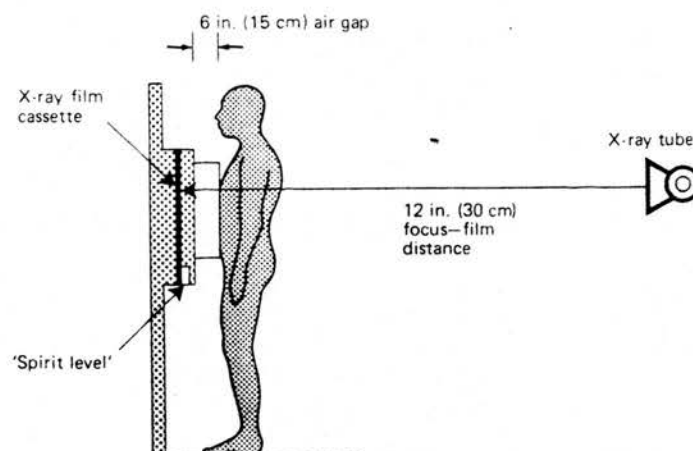


Figure 3.6 Low-dose radiographic technique; the incorporated 'spirit level' acts as a horizontal reference level so that a pelvic tilt can be both identified and measured

of the world. Importantly the films thereby produced are of excellent quality, more than sufficient for scoliosis purposes. In an era when radiation is an emotive word, particularly in the lay population, it is strongly suggested that the low-dose technique be strictly adhered to.

THE SPECTRUM OF SCOLIOSIS IN THE COMMUNITY

Perhaps the biggest problem concerning screening is precisely what idiopathic scoliosis really is. Textbook descriptions of idiopathic scoliosis existing in adolescence refer to a high proportion of female patients, a commoner thoracic curve pattern and almost inevitable progression (James, 1976). In contradistinction, selective community screening harvests enormous numbers of children with small magnitude curves, usually low down in the spine with females not greatly over-represented, in whom only a small fraction show any evidence of progression, more even resolving (Rogala, Drummond and Gurr, 1978; Dickson, 1983). Yet for reasons that are not clear they have all been regarded as examples of idiopathic scoliosis. The Oxford Scoliosis Study Group demonstrated that there are three main types of scoliosis in the community: (1) pelvic tilt scoliosis, (2) spinal scoliosis (a scoliosis that is at least inherent to the spine itself), (3) progressive 'idiopathic' scoliosis (Dickson, 1983). Only two out of 8000 children screened by the Oxford group had an associated congenital spinal anomaly and none appeared to have an associated musculoskeletal condition. In the UK children with such conditions as myelodysplasia or serious neuromuscular disorders tend to be gathered in small pockets in physically handicapped schools, and therefore statistically speaking they scarcely exist in the community's ordinary schools.

Pelvic tilt scoliosis (PTS)

The prevalence rate of scoliosis of all types in the UK, as determined by the visual examination of 10–14-year-old schoolchildren, followed by confirmatory low-dose spinal radiography, is 15%, which agrees well with worldwide figures with the exception of the South African Bantu, whose prevalence rate is about 100 times less (Segil, 1974). Half of these 'positive' children have a scoliosis entirely compensatory to a tilt of the pelvis. The remainder have spinal scoliosis (of whom only one-tenth have the characteristics of idiopathic scoliosis).

In the majority of cases the tilt of the pelvis is due to a minor degree of irrelevant leg length inequality for which treatment had never been sought and which would have remained undetected unless harvested by the screening procedure (*Figure 3.7*). In this situation the leg length inequality produces a hip tilt (a tangential line drawn across the femoral heads is not horizontal) which is translated through the pelvis, providing a pelvic tilt of the same angle. In order to centralize the head over the sacrum, a secondary lumbar scoliosis, or thoracolumbar if it is long enough for the apical vertebra to be at D12 or L1, develops. That such non-structural curves

are associated with some rotation is clearly evidenced in the radiographs and by the fact that they were detected primarily on a forward-bend test.

In one-quarter of cases of pelvic tilt, however, there is no discernible leg-length inequality and the asymmetry occurs in the pelvis itself. This may be an artefact in the coronal plane produced by rotation, possibly occasioned by persistent femoral neck anteversion. In the remaining cases the pelvic tilt is a combination of both a leg-length inequality and pelvic asymmetry. When followed longitudinally these curves show no evidence of progression or regression out of proportion with the

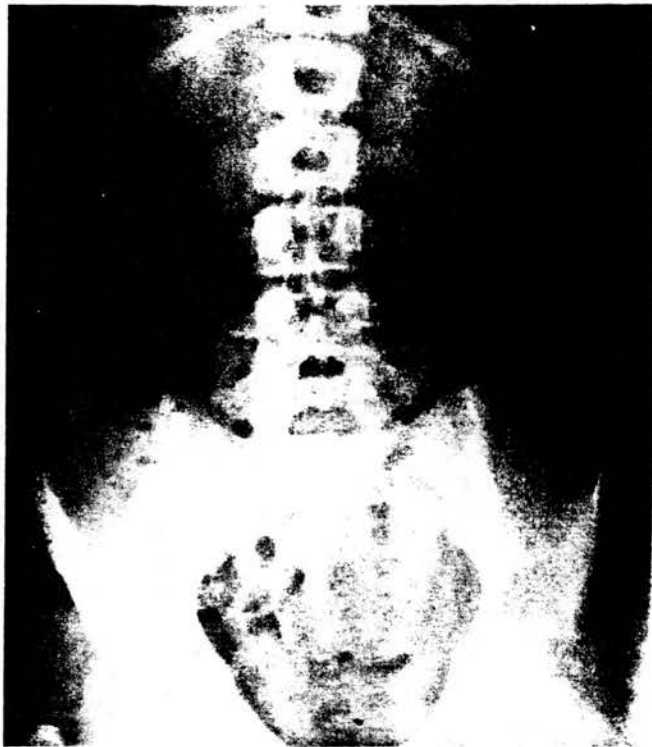


Figure 3.7 Pelvic tilt scoliosis: there is a lumbar curve secondary to a leg length inequality causing a tilted sacrum (For a better quality picture see Publication 4, Figure 2.)

change in leg-length inequality and therefore as a condition it is inconsequential. Its importance lies in its high prevalence rate, which is the chief factor diluting the intensity of screening reports. Furthermore, this condition has an equal sex ratio, curves are low down in the spine, with no preponderance for being to the right or left and with no progression potential even when the leg-length inequality has achieved clinical significance (Papaioannou *et al.*, 1981). These factors go a long way to explain the low female-to-male sex ratio and high proportion of non-progressive lumbar curves which so typify 'schooliosis' (Leatherman, 1982, personal communication).

Table 3.5 Prevalence of all scolioses and proportions with spinal scoliosis (SS) and pelvic tilt scoliosis (PTS)

Curve size (degrees)	Number	Prevalence All (%)	Proportions	
			SS (%)	PTS (%)
<5	241	4.5		
5-9	377	7.1	57	43
10-14	105	2.0	69	31
15-19	32	0.6	75	25
20+	13	0.2	92	8
Total	768	14.4	61	39

As curve magnitude increases, so the proportion with spinal scoliosis reciprocally increases such that if the threshold for the diagnosis of scoliosis were 15° the prevalence rate and dilutional effect of pelvic tilt scoliosis would be 20 times less (*Tables 3.5* and *3.6*). When identified and then eliminated the characteristics of spinal scoliosis become clear and the condition begins to resemble what little we know of idiopathic scoliosis.

Spinal scoliosis

With the elimination of pelvic tilt scoliosis the female-to-male sex ratio rises to 1.6:1 for curves of all sizes, but with increasing curve size the female-to-male sex ratio dramatically increases, being greater than 12:1 for curves measuring 20° or

Table 3.6 Prevalence of scoliosis and sex of children

Curve size (degrees)	All			SS			PTS		
	Female %	Male %	F/M	Female %	Male %	F/M	Female %	Male %	F/M
5-9	7.7	6.6	1.2/1	4.5	3.6	1.3/1	3.1	3.0	1.0/1
10-14	2.7	1.3	2.1/1	2.0	0.8	2.5/1	0.8	0.5	1.6/1
15-19	1.0	0.3	3.3/1	0.7	0.2	3.5/1	0.2	0.1	2.0/1
20+	0.5	0	-	0.5	0	-	0.04	0	-
Total	11.9	8.2	1.5/1	7.7	4.6	1.7/1	4.14	3.6	1.2/1

more (*Tables 3.5* and *3.6*). Curves in the thoracic region are now found to be the commonest, with lumbar curves making up only one-quarter of cases. Left-sided curves are commoner at all sites but while this remains so with the increasing curve magnitude for thoracolumbar and lumbar curves, there is a change to a right-sided preponderance with increasing curve magnitude in the thoracic region (*Tables 3.7* and *3.8*). When thoracic curves are then analysed with respect to the sex of the

Table 3.7 Curve site

Curve size (degrees)	All			SS			PTS		
	Number of curves	Thoracic %	Thoraco- lumbar %	Number of curves	Thoracic %	Thoraco- lumbar %	Number of curves	Thoracic %	Thoraco- lumbar %
5-9	394	24	26	232	40	38	162	0	9
10-14	122	30	23	89	40	26	33	0	15
15-19	34	26	32	26	35	35	8	0	25
20+	17	36	24	16	38	25	1	0	0
Total	567	25	26	363	40	34	204	0	11
									89

Table 3.8 Curve direction

Curve size (degrees)	Thoracic			SS Thoracolumbar			PTS		
	Number of curves	Right %	Left %	Number of curves	Right %	Left %	Number of curves	Right %	Left %
5-9	93	38	62	89	33	67	162	50	50
10-14	36	47	53	23	30	70	33	52	48
15-19	9	33	67	9	22	78	8	25	75
20+	6	67	33	4	75	25	1	100	-
Total	144	41	59	125	34	66	204	50	50

child, the overall left-sided predominance is due to boys who have more left-sided curves which remain left-sided with increasing curve magnitude. The bigger the curve magnitude in girls, the greater the proportion with right-sided curves.

Thus with increasing curve magnitude traditional texts are proved to be correct, with girls greatly outnumbering boys and right thoracic curves becoming more obvious. What, then, is idiopathic scoliosis? Cross-sectional analyses with reference to curve magnitude certainly imply that idiopathic scoliosis is that small fraction of spinal scoliosis which has progressed to attain a bigger curve magnitude. Former definitions of idiopathic scoliosis are poor and do not take into account progression which so typifies the clinical syndrome. Even having eliminated the proportion of the community with pelvic tilt scoliosis cannot mean that the rest all have idiopathic scoliosis, when we know that if the screening method were more sensitive (Inkster, 1953; Adair, Van Wijk and Armstrong, 1977; Burwell *et al.*, 1982) substantially more if not all children would show evidence of coronal plane asymmetry of the spine and when we also know that only three per 1000 of the Oxford series achieved a curve magnitude of 20°. Little more can be achieved with a cross-sectional analysis and it is therefore necessary to look at what happens when these children are followed longitudinally as a cohort to determine what effect, if any, growth has.

Progressive 'idiopathic' scoliosis

When children with spinal scoliosis measuring 10° or more are followed longitudinally as a cohort the situation becomes clearer. Two-thirds do not change with regard to curve magnitude, two-fifths regress (a reduction in curve magnitude by 5° or more), and one-tenth progress (increase in curve magnitude by 5° or more) (Figure 3.8; Table 3.9). The vast majority of spinal scolioses therefore have a very benign course, remaining static or even resolving. Those that progress must represent true idiopathic scoliosis, at least as far as we currently know it. This is where statistical analysis is essential in order to determine what features indicate progression potential which are not the result of chance. It is interesting that there is no particular tendency for thoracic, thoracolumbar or lumbar curves to either progress or for that matter regress. Nor is progression a particular feature of females when curves in all sites are considered. However, when thoracic curves are analysed, girls with right-sided curves have a significant tendency to progress while boys and left-sided curves have a significant tendency to regress. This confirms the inferences of the cross-sectional analysis and provides useful guidelines as regards the follow-up of children detected as having a scoliosis by screening. Clearly girls with right-sided thoracic curves are particularly vulnerable and demand the closest scrutiny, while the opposite can be said for boys with left-sided curves.

Curve direction

It is often wondered why in idiopathic scoliosis of clinical significance thoracic curves tend to go to the right, while lumbar curves tend to go to the left. With increasing curve magnitude these trends do become more obvious, such that eight times more curves in the thoracic region of clinical significance are convex to the

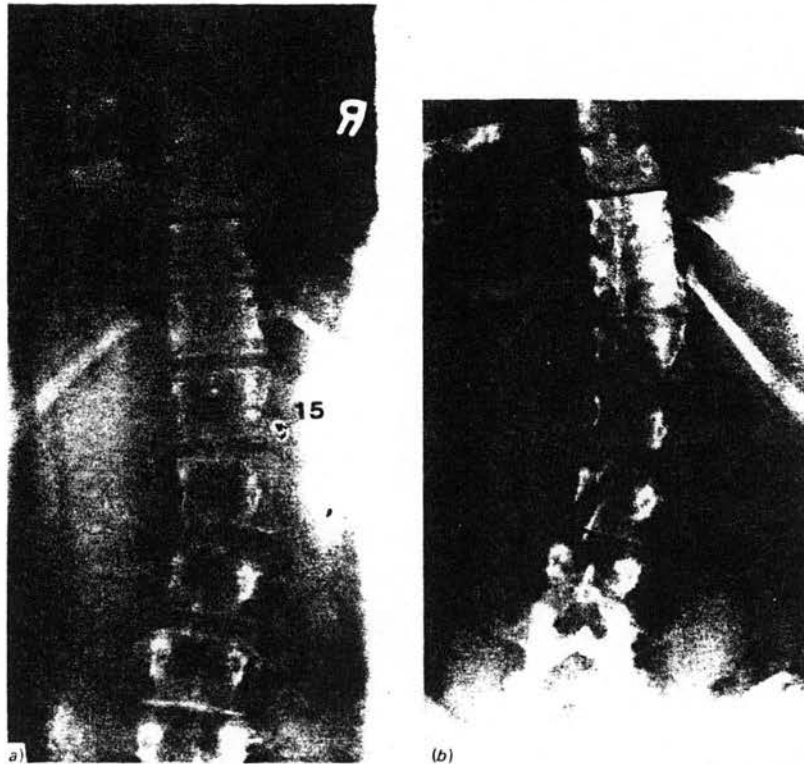


Figure 3.8 Progressive idiopathic scoliosis: (a) the curve measures 15° initially; (b) 1 year later the curve measures 23° (from Dickson *et al.*, 1980, courtesy of the Editor and Publishers, *British Medical Journal*) (For a better quality picture see Publication 1, Figure 3.)

right. However, the smallest magnitude curves are in general randomly directed and therefore the question that should be asked is not why bigger curves in particular sites go to a particular direction but why they do not progress in the other direction. Rather than something pushing them in one direction it is more logical to consider something preventing them from going in the other.

There is strong evidence that the thoracic vertebral bodies are not symmetrical either in the coronal or transverse planes either during growth or any time afterwards (Inkster, 1953). The fourth to the eighth thoracic vertebral bodies are constantly grooved on the left side by the developing thoracic aorta and this is still eminently visible in adult autopsy specimens. With such a potent force pulsating 72

Table 3.9 Fate of curves measuring 10° or more

	Progress (%)	Regress (%)	Static (%)
Girls	16	21	63
Boys	5	33	62

times per minute after maturity, and much more frequently before, it could be speculated that this provides the physical barrier for the prevention of randomly occurring left-sided curves from progression. While it can also be speculated that patients with dextrocardia might be expected to have a higher prevalence of left-sided curves, the low prevalence rate of this abnormality hinders statistical confirmation. However, the shape of the lateral profile of the spine in idiopathic scoliosis has another important effect (*see* Chapter 1).

The effect of growth

Growth is manifestly an important environment in which the spinal deformity first appears and in a small minority subsequently progresses. It has already been suggested that it is the overall environment of growth and not growth velocity that is more relevant (Duthie, 1971), yet most attention has been directed towards measuring when a child has stopped growing (Risser, 1958) rather than trying to define accurately the biological age of the child, which is so much more important than chronological age (Tanner, Whitehouse and Takaishi, 1966). It has been suggested that girls with idiopathic scoliosis are taller than their straightbacked counterparts and more advanced earlier in adolescence and retarded later (Willner, 1974; Nordwall and Willner, 1975; Low *et al.*, 1978). Control heights were derived from data pertaining to earlier generations against which children today would appear taller, whether or not scoliosis was associated with increased height. Growth studies must therefore make use of local contemporaneous controls of not dissimilar social class and for that matter use a method of determining skeletal age which is more up to date and more accurate. Although not perfect by any means, the second method of Tanner and Whitehouse is a more accurate determinant and rapidly provides skeletal age to one-tenth of a year.

When studies using these up-to-date standards are performed, children with idiopathic scoliosis show no difference whatsoever from normal children with regard to standing height, sitting height, puberty rating or skeletal age (Dickson and Sevitt, 1982). This lends further support to the concept that it is the overall environment of growth that is important and not the specificities. Since it was first reported that some cases of idiopathic scoliosis progress until well into the 20s, seemingly those associated with repeated pregnancies, although there is no statistical basis for this statement, a 'soft tissue factor' can be speculated. It ought not to be forgotten that the spine may still be growing, whatever else the soft tissues are doing.

Longitudinal growth studies have, however, shown one noticeable trend, and that is that children with idiopathic scoliosis with bigger curves are significantly taller than those with smaller curves, but they showed no evidence of growing faster (Dickson and Sevitt, 1982). This observation was confirmed in a cross-sectional analysis of southern Chinese girls in which those with idiopathic scoliosis were shown to be taller than contemporaneous controls (Leong *et al.*, 1982). However, if this selected group of relatively severe curves were to be compared with smaller curves a similar trend would be found, lending support to the concept that stature rather than growth is important for progression. This evidence of 'genetic tall

stature' (they come from big families) is entirely explicable on the basis of uncoiling of the lateral profile of children with idiopathic scoliosis (see Chapter 1). This goes a long way towards explaining the familial trends observed with regard to idiopathic scoliosis (Wynne-Davies, 1968) in that it is in these families with a flatter lateral profile that one would expect to find an increased prevalence of idiopathic scoliosis. There is abundant evidence that the whole phenomenon of growth and shape is very strictly controlled genetically, with families sharing the same times and patterns for menarche, growth velocity, maturity, etc. (Bolk, 1923; Boas, 1932; Hewitt, 1957). It is more likely therefore that the familial nature of idiopathic scoliosis reflects nothing more than the familial genes for growth and lateral profile shape.

SUMMARY

In the last 20 years millions of children have been screened in much of the world for a condition about which the natural history is still far from clear. What is being screened for is some rotation with or without coronal plane asymmetry and this is probably normal in growing children. It is as well that the visual screening examination is so crude that it only detects 15% of the population; 10–14-year-olds have been selected as being particularly at risk but the necessary epidemiological research to support this is lacking. The consequences of untreated scoliosis are certainly devastating but apply only to curves of much earlier onset. Adolescent onset idiopathic scoliosis is a relatively benign condition, and with only a minority of curves of 20° or more actually progressing this has the effect of flattering the efficacy of conservative treatment, which is almost certainly overprescribed.

The presence of inconsequential pelvic tilt scoliosis in half of those harvested makes screening reports difficult to evaluate. Workers are urged to seek epidemiological assistance before embarking upon screening, which should in any event be conducted with natural history and not prevention primarily in mind. In any screening programme radiographic analysis is necessary but the X-ray techniques involved must be of a low-dose variety so that the great majority who do not have idiopathic scoliosis can be protected accordingly.

References

- Abbott, E. V. (1977) Screening for scoliosis: a worthwhile preventive measure. *Canadian Journal of Public Health*, **68**, 22–25
- Adair, I. V., Van Wijk, M. C. and Armstrong, G. W. D. (1977) Moiré topography in scoliosis screening. *Clinical Orthopaedics*, **129**, 165–171
- Ardran, G. M., Coates, R., Dickson, R. A., Dixon-Brown, A. and Harding, F. M. (1980) Assessment of scoliosis in children. Low-dose radiographic technique. *British Journal of Radiology*, **53**, 146–147
- Ascani, E., Salsano, V. and Giglio, G. (1977) The incidence and early detection of spinal deformities. *Italian Journal of Orthopaedics and Trauma*, **3**, 111–117
- Bellyei, A., Czeizel, A., Barta, O., Magda, T. and Molnar, L. (1977) Prevalence of adolescent idiopathic scoliosis in Hungary. *Acta Orthopaedica Scandinavica*, **48**, 177–180

- Belstead, J. S. and Edgar, M. A. (1978) Early detection of scoliosis. *British Medical Journal*, **2**, 937-938
- Bengtsson, G., Fallstrom, K., Jansson, B. and Nachemson, A. (1974) A psychological and psychiatric investigation of the adjustment of female scoliosis patients. *Acta Psychiatrica Scandinavica*, **50**, 50-59
- Boas, F. (1932) Studies in growth. *Human Biology*, **4**, 307-350
- Bolk, L. (1923) The menarche in Dutch women and its precipitated appearance in the youngest generation. *Proceedings of the Academy of Science, Amsterdam*, **26**, 250
- Brooks, H. L., Azen, S. P., Gerberg, E., Brooks, R. and Chan, L. (1975) Scoliosis: a prospective epidemiological study. *Journal of Bone and Joint Surgery*, **57A**, 968-972
- Burwell, R. G., James, N. J., Johnson, F. and Webb, J. K. (1982) The rib hump score: a guide to referral and prognosis? *Journal of Bone and Joint Surgery*, **64B**, 248
- Carr, W. A., Moe, J. H., Winter, R. B. and Lonstein, J. E. (1980) Treatment of idiopathic scoliosis in the Milwaukee brace. Long-term results. *Journal of Bone and Joint Surgery*, **62A**, 599-612
- Collis, D. K. and Ponseti, I. V. (1969) Long-term follow-up of patients with idiopathic scoliosis not treated surgically. *Journal of Bone and Joint Surgery*, **51A**, 425-445
- Commission on Chronic Illness (1957) *Chronic Illness in the United States*, Vol. I. Cambridge, Mass.: Harvard University Press
- Cronis, A. and Russell, A. Y. (1965) Orthopaedic screening of children in Delaware public schools. *Delaware Medical Journal*, **37**, 89-92
- Dahlberg, L. and Nachemson, A. (1977) The economic aspects of scoliosis treatment. In *Scoliosis. Proceedings of a Fifth Symposium*, edited by P. A. Zorab, 73-101. London: Academic Press
- DeSmet, A. A., Fritz, S. L. and Asher, M. A. (1981) A method for minimizing the radiation exposure from scoliosis radiographs. *Journal of Bone and Joint Surgery*, **63A**, 156-161
- Dickson, R. A. (1983) Scoliosis in the community. *British Medical Journal*, **286**, 615-618
- Dickson, R. A. and Leatherman, K. D. (1976) Spinal deformity in adults - changing concepts. *Journal of Bone and Joint Surgery*, **58A**, 729
- Dickson, R. A. and Leatherman, K. D. (1978) Cotrel traction, exercises, casting in the treatment of idiopathic scoliosis. *Acta Orthopaedica Scandinavica*, **49**, 46-48
- Dickson, R. A. and Sevitt, E. A. (1982) Growth and idiopathic scoliosis: a longitudinal cohort study. *Journal of Bone and Joint Surgery*, **64B**, 385
- Dickson, R. A., Stamper, P., Sharp, A. M. and Harker, P. (1980) School screening for scoliosis: cohort study of clinical course. *British Medical Journal*, **281**, 265-267
- Duthie, R. B. (1971) Growth and its relation to bone deformities. In *Scoliosis and Growth, Proceedings of a Third Symposium*, edited by P. A. Zorab. Edinburgh: Churchill Livingstone
- Flynn, J. C., Riddick, M. F. and Keller, T. L. (1977) Screening for scoliosis in Florida schools. *Journal of the Florida Medical Association*, **64**, 159-161

- Golomb, M. and Taylor, T. F. (1975) Screening adolescent schoolchildren for scoliosis. *Medical Journal of Australia*, **2**, 761-762
- Gray, J. E., Hoffman, A. D. and Peterson, H. A. (1983) Reduction of radiation exposure during radiography for scoliosis. *Journal of Bone and Joint Surgery*, **64A**, 5-12
- Greulich, W. W. and Pyle, S. I. (1959) *Radiographic Atlas of Skeletal Development of the Hand and Wrist*. 2nd edition. Stanford: Stanford University Press, and London: Oxford University Press
- Hassan, I. and Bjerkreim, I. (1983) Progression in idiopathic scoliosis after conservative treatment. *Acta Orthopaedica Scandinavica*, **54**, 88-90
- Hewitt, D. (1957) Some familial correlations in height, weight and skeletal maturity. *Annals of Human Genetics*, **22**, 26-35
- Inkster, R. G. (1953) Osteology. In *Cunningham's Textbook of Anatomy*, edited by J. C. Brash, 9th edition, 122-137. London: Oxford Medical Publications
- Inoue, S. (1978) Moiré topography for the early detection and postoperative evaluation of scoliosis. Paper presented to the American Orthopaedic Research Society
- James, J. I. P. (1976) Idiopathic scoliosis. In *Scoliosis*, 2nd edition. Edinburgh: Churchill Livingstone
- Kane, W. J. and Moe, J. H. (1970) A scoliosis-prevalence survey in Minnesota. *Clinical Orthopaedics*, **69**, 216-218
- Kostuik, J. P., Israel, J. and Hall, J. E. (1973) Scoliosis surgery in adults. *Clinical Orthopaedics*, **93**, 225-234
- Leaver, J. M., Alvik, A. and Warren, M. D. (1983) Prescriptive screening for idiopathic scoliosis: a review of the evidence. *International Journal of Epidemiology*, **11**, 101-111
- Leong, J. C. Y., Low, W. D., Mok, C. K., Kung, L. S. and Yau, A. C. M. C. (1982) Linear growth in southern Chinese female patients with adolescent idiopathic scoliosis. *Spine*, **7**, 471-475
- Lonstein, J. E. (1977) Screening for spinal deformities in Minnesota schools. *Clinical Orthopaedics*, **126**, 33-42
- Low, W. D., Mok, C. K., Leong, J. C. Y., Yau, A. C. M. C. and Lisowski, F. P. (1978) The development of southern Chinese girls with adolescent idiopathic scoliosis. *Spine*, **3**, 152-156
- Nachemson, A. (1968) A long-term follow-up study of non-treated scoliosis. *Acta Orthopaedica Scandinavica*, **39**, 466-476
- Newman, D. C. and DeWald, R. L. (1977) School screening for scoliosis. *Illinois Medical Journal*, **152**, 31-34
- Nilsson, U. K. and Lundgren, K. D. (1968) Long-term prognosis in idiopathic scoliosis. *Acta Orthopaedica Scandinavica*, **39**, 456-465
- Nordwall, A. and Willner, S. (1975) A study of skeletal age and height in girls with idiopathic scoliosis. *Clinical Orthopaedics*, **110**, 6-10
- O'Brien, J. P. and van Akkerveken, P. F. (1977) School screening for scoliosis. Results of a pilot study. *The Practitioner*, **219**, 739-742
- Papaioannou, M., Stokes, I. A. F., Stamper, P., Dickson, R. A. and Kenwright, J. (1981) Spine asymmetry and leg length inequality. *Journal of Bone and Joint Surgery*, **63B**, 641

- Petrie, A. (1978) *Lecture Notes on Medical Statistics*, 155. Oxford: Blackwell Scientific Publications
- Ponder, C. R., Dickson, J. H., Harrington, P. R. and Erwin, W. D. (1975) Results of Harrington instrumentation and fusion in the adult idiopathic scoliosis patient. *Journal of Bone and Joint Surgery*, **57A**, 797-801
- Ponseti, I. V. and Friedman, B. (1950) Prognosis in idiopathic scoliosis. *Journal of Bone and Joint Surgery*, **32A**, 381-395
- Reid, L. (1971) Lung growth. In *Scoliosis and Growth, Proceedings of a Third Symposium*, edited by P. A. Zorab. Edinburgh: Churchill Livingstone
- Risser, J. C. (1958) The iliac apophysis: an invaluable sign in the management of scoliosis. *Clinical Orthopaedics*, **11**, 111
- Rogala, E. G., Drummond, D. S. and Gurr, J. (1978) Scoliosis: incidence and natural history. *Journal of Bone and Joint Surgery*, **60A**, 173-176
- Sabatier (1777) *Traite complète d'anatomie*. Paris. Quoted by A. Farkas (1941). Physiological scoliosis. *Journal of Bone and Joint Surgery*, **23**, 607-627
- Segil, C. M. (1974) The incidence of scoliosis in the Bantu and white population groups in Johannesburg. *Journal of Bone and Joint Surgery*, **56B**, 393
- Sells, C. J. and May, E. A. (1974) Scoliosis screening in public schools. *American Journal of Nursing*, **74**, 60-62
- Shands, A. R. and Eisenberg, H. B. (1955) The incidence of scoliosis in the state of Delaware. A study of 50 000 microfilms of the chest made during a survey for tuberculosis. *Journal of Bone and Joint Surgery*, **37A**, 1243-1249
- Smyrnis, P. N., Valavanis, J., Alexopoulos, A., Siderakis, G. and Giannestras, N. J. (1979) School screening for scoliosis in Athens. *Journal of Bone and Joint Surgery*, **61B**, 215-217
- Span, Y., Robin, G. and Makin, M. (1976) The incidence of scoliosis in schoolchildren in Jerusalem. *Journal of Bone and Joint Surgery*, **58B**, 379
- Strayer, L. M. (1973) The incidence of scoliosis in the post-partum female on Cape Cod. *Journal of Bone and Joint Surgery*, **55A**, 436
- Tanner, J. M. (1962) *Growth at Adolescence*, 2nd edition. Oxford: Blackwell Scientific Publications
- Tanner, J. M., Whitehouse, R. H. and Takaishi, M. (1966) Standards from birth to maturity for height, weight, height velocity and weight velocity. *Archives of Disease in Childhood*, **41**, 613-635
- Torell, G., Nordwall, A. and Nachemson, A. (1981) The changing pattern of scoliosis treatment due to effective screening. *Journal of Bone and Joint Surgery*, **63A**, 337-341
- Whitby, L. G. (1974) Screening for disease. Definitions and criteria. *Lancet*, **2**, 819-822
- White, A. A. (1971) Kinematics of the normal spine as related to scoliosis. *Journal of Biomechanics*, **4**, 405-411
- Willner, S. (1974) A study of growth in girls with adolescent idiopathic structural scoliosis. *Clinical Orthopaedics*, **101**, 129-135
- Wynne-Davies, R. (1968) Familial (idiopathic) scoliosis. A family survey. *Journal of Bone and Joint Surgery*, **50B**, 24-30

SCREENING FOR SCOLIOSIS : THE PROBLEM OF ARM LENGTH.

Stirling AJ, Smith RM, Dickson RA.

British Medical Journal; 292: 1305-1306, 1986.

This study involving 113 school-children demonstrated the ability of small differences in arm length to produce variable degrees of rib hump on forward bending. Forward bending with the fingertips together could produce evidence of a spinal deformity, because of unrecognised arm length inequality, when the spine was not in fact deformed. This finding invalidated or diluted the results of many other scoliosis screening programmes which did not pay regard to this problem, and a standard forward bending test was described which obviated concern about arm length inequality.

Screening for scoliosis: the problem of arm length

The forward bend test is an integral part of the assessment of scoliosis, whether for screening purposes or in the established condition. The introduction of the scoliometer, an inclinometer placed over the area of maximal deformity, has provided a rapid method of quantifying the rib or lumbar hump, and a recommended threshold for further follow up or orthopaedic assessment has been suggested.¹ An increased prevalence of arm length asymmetry exists in adolescent idiopathic scoliosis.² The influence of this recognised variable on forward bend assessment has been ignored in many papers reporting results from scoliosis screening programmes. Some reports recommend that the arms hang freely (position A),³ others that the hands are placed together with fingertips apposed (position B),⁴ and in many no accurate description of the position used is given. We examined the effect of different arm positions on scoliometer readings in 113 schoolchildren.

Patients, methods, and results

One hundred and thirteen schoolchildren (41 girls and 72 boys) aged 13 or 14 were examined by two independent observers. Arm lengths (from acromion to tip of middle finger) were measured to the nearest mm. Scoliometer readings of the magnitude and direction of any asymmetry were taken at the level of the spinous processes of the fourth and eighth thoracic vertebrae in positions A and B (described above) and also with the fingertips of the left hand apposed to the proximal finger crease of the right hand (position C) and vice versa (position D).

Both observers showed the right arm to be longer than the left in the group overall and in girls. In boys, however, the right arm was longer when recorded by one observer and the left when recorded by the other (see table). The maximum difference observed in arm length was 24 mm.

Mean (SEM) scoliometer readings (in scoliometer degrees). Positive values indicate a left sided rib hump, negative values a right sided rib hump

Position	Thoracic vertebrae	Girls	Boys
A	T4	-0.27 (0.27)	-0.42 (0.16)
	T8	0.00 (0.32)	-0.06 (0.18)
B	T4	-0.28 (0.26)	-0.45 (0.17)
	T8	-0.17 (0.34)	0.02 (0.17)
C	T4	-5.60 (0.28)	-6.10 (0.25)
	T8	-3.06 (0.31)	-2.99 (0.22)
D	T4	5.28 (0.30)	5.40 (0.23)
	T8	2.88 (0.33)	3.08 (0.20)

There was a highly significant correlation in the scoliometer readings recorded by the two observers in all positions ($p < 0.0001$), and thus mean scoliometer readings were subsequently used. The table gives the mean scoliometer reading in each position. A significant correlation (Pearson product-moment) between the difference in arm length and the change in scoliometer reading between position A and B was found only by one observer, at T8 ($p < 0.02$). Positive correlations were found, however, between these two variables for both observers for the whole group and for the girls alone; all but one of the coefficients for the boys were also positive. When both observers' results are considered at the T4 and T8 levels by sex the overall correlation is significant (sign test; $p < 0.05$). The consistent production of a noticeable rib hump of roughly 6° at T4 on the same side as the "longer" arm in positions C and D was seen clearly. This finding was constant for both observers and it reversed to the opposite side when changing from position C to D. The scoliometer readings at T8 in positions C and D showed a similar hump, but of only 3° . Again this reversed consistently between positions C and D.

Comment

This study shows that true arm length inequality has an important influence on the perceived rib hump when a forward bend position with the hands apposed is used. Furthermore, if the same position is used and an artificial arm length inequality of roughly 5 cm made an apparent and reproducible rib hump of 6° at T4 and 3° at T8 results. Exaggeration of the apparent deformity will be proportional to the magnitude of arm length inequality present, and this may be sufficient to influence the scoliometer reading and push a child over the threshold for referral. The results of screening programmes will accordingly be inaccurate. This influence may be eliminated by adopting a standard position for the forward bend test with the arms hanging freely.

- 1 Bunnell WP. An objective criterion for scoliosis screening. *J Bone Joint Surg* 1984;66A:1381-7.
- 2 Burwell RG, Dangerfield PH, Vernon CL. Anthropometry and scoliosis. In: Zorab PA, ed. *Scoliosis*. London: Academic Press 1977:123-63.
- 3 Lonstein JE. Screening for spinal deformities in Minnesota schools. *Clin Orthop* 1977;126:33-42.
- 4 Ascani E, Salsano V, Giglio G. Incidence and early detection of spinal deformities. *Ital J Orthop Traumatol* 1977;3:111-7.
- 5 Wynne-Davies R. Familial (idiopathic) scoliosis. *J Bone Joint Surg* 1968;50B:24-30.

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IDIOPATHIC SCOLIOSIS : THE LEEDS EPIDEMIOLOGICAL SURVEY.

Stirling AJ, Dickson RA.

Journal of Bone and Joint Surgery; 68B: 497, 1986.

Screening programmes and epidemiological surveys hitherto had focused on adolescents (children between the ages of 9 or 10 and 14), for no other reason than that the prevalence rate was known to be high during adolescence. Moreover, only relatively small numbers had been studied. With the my move from Oxford to Leeds the opportunity to study much larger numbers (16 000 school-children) including a younger age group (6 to 13 year olds inclusive) was afforded by a generous five year programme grant from the Medical Research Council. I was also interested in the sagittal profile of the spine (see later under Pathogenesis section), flattening of which in the thoracic region appeared to be intrinsic to the deformity of idiopathic scoliosis. These 16 000 children were duly screened during one year and a cohort of 980 with evidence of trunk asymmetry were then assessed by the low dose radiographic technique as regards their spinal shape. A host of other variables including stature, growth velocity and bone age by the Tanner and Whitehouse second method (TW2 bone age) were also recorded. This cohort has been followed longitudinally, the variables being repeated each year. This particular paper refers to the first year's cross-sectional analysis and demonstrated a rising prevalence rate in both sexes through the ages with a significant number of 6 year olds with an appreciable deformity. This particular abstract summarises the first year of the study.

Idiopathic scoliosis: the Leeds epidemiological survey—*A. J. Stirling and Professor R. A. Dickson* (Leeds) stated that the natural history of idiopathic scoliosis was still unclear, particularly in younger children in whom the condition was more serious. Accordingly, in an epidemiological survey of 16 000 schoolchildren, 2000 at each year of age from 6 to 13 were included. A spinal deformity was recorded in 980 (5.7%) on the basis of physical signs or a scoliometer reading in excess of 5°. Low-dose postero-anterior spinal radiographs were obtained and curve characteristics recorded.

The prevalence rate of idiopathic scoliosis measuring 5° or more was 3.2%, being 3.6% for girls and 2.8% for boys. For both sexes there was a significant trend for the prevalence rate to rise with age (0.8% and 2.7% for boys and girls aged six respectively, to 7.5% and 8.1% for boys and girls aged 13 respectively). When prevalence rate was analysed with respect to curve size, there were significantly more girls than boys at all ages with bigger curves.

These younger age groups had hitherto not been included in such surveys and the much higher than expected prevalence rate of idiopathic scoliosis in six-year-olds (50% of the figure for 12-year-old girls, and 25% of the figure for 12-year-old boys) had important implications. They suggested that a substantial number of so-called adolescent-onset idiopathic scoliosis cases truly commence at a much earlier age.

THE FIRST YEAR'S FOLLOW-UP OF THE MRC SCOLIOSIS SCREENING PROGRAMME.

Whitaker IA, Stirling AJ, Dickson RA.

Journal of Bone and Joint Surgery; 69B: 851, 1987.

The classification of spinal deformities in the community was extended to include four types, from inconsequential pelvic tilt scoliosis at one end of the spectrum to definite idiopathic cases at the other (concordant rotation refers to rotation of the posterior elements to the curve concavity). Idiopathic cases were noted to have the greatest progression potential and the most important variable in discriminate analysis of progression was found to be flattening of the lateral spinal profile (the degree of thoracic lordosis).

The first year's follow-up of the MRC scoliosis screening programme – *I. A. Whitaker, A. J. Stirling and Professor R. A. Dickson* (Leeds) reported on a cohort group of 960 children aged between 6 and 16 years who were found to have trunk asymmetry on forward bending. The children were examined and had two standardised postero-anterior radiographs of the spine at an interval of one year: these were assessed for spinal deformity in the coronal plane by the Cobb method and for evidence of spinal rotation using the Perdriolle method. On the basis of these measurements children with Cobb angles of 5° or more were placed into four groups: (1) idiopathic – those with coronal deformity and concordant rotation of the apex; (2) non-classifiable with coronal deformity but no rotation at the apex; (3) rotated kyphoses with coronal deformity and discordant rotation at the apex; and (4) pelvic tilt scoliosis.

Using these two serial measurements, it was possible to identify a group in whom the Cobb angle had increased by 5° or more over the period of a year. Analysis showed a definite trend for children in Group 1 (idiopathic curves) to have a greater propensity towards progression.

THE PATHOGENESIS OF IDIOPATHIC SCOLIOSIS - BI-PLANAR SPINAL ASYMMETRY.

Dickson RA, Lawton JO, Archer IA, Butt WP.

Journal of Bone and Joint Surgery; 66B: 8-15, 1984.

This clinical, cadaveric, biomechanical and radiological investigation of the pathogenesis of idiopathic scoliosis demonstrated that idiopathic thoracic scoliosis was associated with a primary abnormality of spinal shape in the sagittal plane in the nature of a lordosis at the apex of the deformity. Although this possible mechanism had been alluded to in the past, this paper first produced scientific confirmation that a scoliosis (lateral curvature of the spine) was secondary to a primary abnormality in the sagittal plane. Moreover, this primary idiopathic abnormality explained much of the clinical behaviour of structural spinal deformities.

THE PATHOGENESIS OF IDIOPATHIC SCOLIOSIS

BIPLANAR SPINAL ASYMMETRY

R. A. DICKSON, J. O. LAWTON, I. A. ARCHER, W. P. BUTT

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A clinical, cadaveric, biomechanical and radiological investigation of the pathogenesis of idiopathic scoliosis indicates that biplanar asymmetry is the essential lesion. Many normal children have coronal plane asymmetry (an inconsequential lateral curvature of the spine), and certainly all have vertebral body asymmetry in the transverse plane, but when median plane asymmetry (flattening or more usually reversal of the normal thoracic kyphosis at the apex of the scoliosis) is superimposed during growth, a progressive idiopathic scoliosis occurs. Idiopathic kyphoscoliosis cannot and does not exist, from the mildest cases in the community to the most severe cases in pathology museums. Median plane asymmetry is crucial for progression and the lateral profile of the spine must be carefully scrutinised. Increased anterior vertebral height at the apex of the curve with posterior end-plate irregularity characterises the median plane asymmetry and suggests that idiopathic scoliosis is the reverse of Scheuermann's disease.

Idiopathic scoliosis is a lateral curvature of the spine in the absence of any relevant congenital spinal anomaly or associated musculoskeletal condition. Rotation and progression potential both characterise the clinical deformity. Most workers favour a neuromuscular basis for the condition although growth and genetic factors may be important. Early promising electromyographic findings (Lerique and LeCoeur 1951) have either been invalidated (Alexander and Season 1978) or shown to be secondary to the spinal deformity (Trontelj, Pečak and Dimitrijević 1979). In addition, structural abnormalities of the spinal musculature (Kaneko 1968) have also been shown to be secondary findings (Khosla *et al.* 1980) and equilibrial dysfunction (Yamada *et al.* 1969) is more likely to be a feedback from the deformed spine (Sahlstrand and Petruson 1979). The increased prevalence of idiopathic scoliosis in families (Staub 1922) appears to reflect a growth pattern which families also share (Boas 1932).

"Normal" children have neither straight nor symmetrical spines (Dickson 1983). Fifteen per cent of children show evidence of a lateral curvature on a crude visual test and as many as 30 per cent with a more accurate screening method (Burwell *et al.* 1982). Indeed, anatomists two centuries ago clearly demonstrated that everyone had a scoliosis, albeit of small magnitude (Sabatier 1777). However, only two to five per thousand

have curves measuring 20 degrees or more and therefore clearly another important factor or factors must exist before an unimportant curvature becomes a progressive rotational idiopathic scoliosis.

It has long been known that patients with idiopathic thoracic curves have median (sagittal) plane asymmetry in the nature of a lordosis at the curve apex (Adams 1865) and that the thoracic kyphosis in normal children is considerably reduced in early adolescence (Willner 1981). Furthermore, a combination of lordosis and asymmetry of the spine in the coronal plane has recently been shown to produce "idiopathic" scoliosis experimentally (Dickson *et al.* 1983). This present clinical, cadaveric, biomechanical and radiological study indicates that biplanar spinal asymmetry is the essential lesion of idiopathic scoliosis and adequately explains its clinical behaviour.

MATERIALS AND METHODS

Clinical investigation. Seventy patients with idiopathic thoracic scoliosis were studied: there were 55 females and 15 males and their mean age was 14.2 years (range 7 to 26). All 70 were studied on presentation, and anteroposterior and lateral radiographs were taken before the spinal shape could have been affected by any form of treatment. The anteroposterior radiographs were AP projections of the patient and not of the apex of the rotated curve (Stagnara *et al.* 1975). Fifty-five lateral films also were laterals of the patient, but the 15 most recent lateral films were true laterals of the apex of the rotated curve. From the anteroposterior radiographs the magnitude of the scoliotic curve was measured using Whittle's adaptation of Cobb's method (Whittle and Evans 1979); and from the lateral radiographs both the

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angle of the overall thoracic kyphosis and the angle subtended by the vertebrae at the apex of the scoliosis were similarly recorded. Five children with idiopathic scoliosis were also subjected to computerised axial tomography in order to determine the degree and pattern of rotation of their curves.

Cadaveric analysis. Eleven cadaveric spines with idiopathic scoliosis were borrowed from the Museum of the Royal College of Surgeons in Edinburgh by courtesy of Professor D. C. Meekie. Four were natural specimens and in seven the axial skeleton was reconstructed using artificial material for discs and ligaments. All were subjected to standardised radiography and photography. Anteroposterior and lateral films and photographs were taken of the specimens and then of the apex of the curve. From these films the angles of the scoliosis and the lateral profile of the spine were measured. The four natural specimens were also subjected to computerised tomography in the transverse axial and longitudinal planes.

Biomechanical studies. A simplified theoretical model of a spine with biplanar asymmetry was constructed (Fig. 1), with the important forces and their directions inserted. Then, using fresh natural spines, removed at necropsy from 1400-gram New Zealand white rabbits, the behav-



Fig. 2

Fig. 3

Figure 2—True lateral projection of the apex of an idiopathic thoracic curve. There is a mild lordosis and the apical vertebrae are distinctly wedged, the anterior vertebral height being longer than the posterior with end-plate irregularity. Figure 3—Lateral projection of the apex of a Scheuermann's kyphosis. Again there is vertebral wedging and end-plate irregularity, but exactly the opposite of Figure 2: here the anterior vertebral body height is shorter than the posterior.

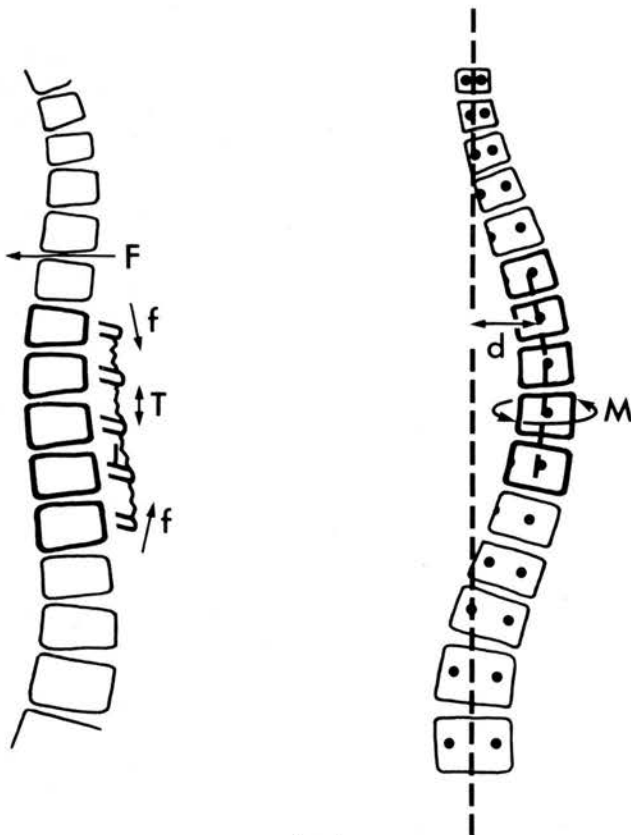


Fig. 1

Simplified theoretical model of biplanar spinal asymmetry. Lateral profile with lower thoracic lordosis (left); anteroposterior view of coronal plane asymmetry (right). $M \propto f \cdot d$, where F =force of forward flexion; T =tightening of posterior structures; f =reactive force; d =distance of coronal plane asymmetry from midline; and M =spinning moment.

our of the spine in relation to the theoretical model was investigated. Specimens were flexed forward to and beyond the limits of normal movement; then, after the creation of a short-segment biplanar asymmetry in the lower thoracic region using compression wiring of the spinous processes, forward flexion was repeated. Sample radiographs were taken through and beyond the normal limits of spinal flexion.

RESULTS

Clinical investigation. The mean Cobb angle of the scoliosis in the 70 patients was 39 degrees and the overall angle of thoracic kyphosis was 20 degrees. However, when the lateral profile at the apex of the scoliosis was measured, all showed evidence of median plane asymmetry. There was a mean lordosis of three degrees at the apex of the curve which involved only the apical two or three segments. More specifically, 75 per cent of these apical lateral profiles were lordotic, 24 per cent straight; one per cent were kyphotic, but of a radius much greater than that of the kyphosis above. There was no kyphosis below the apex, indicating that the lordotic area was an abnormal continuation upwards of the lumbar lordosis. The apical one or two vertebrae were observed to be distinctly wedged, the anterior vertebral body height being greater than the posterior height, often with end-plate irregularity posteriorly (Figs 2 and 3).

There was a significant correlation between the Cobb angle of the scoliosis and that of the overall kyphosis ($P < 0.01$); and the apical lateral Cobb angle was significantly negatively correlated with the angle of

overall kyphosis ($P < 0.001$). The position of the median plane asymmetry correlated with the apex of the scoliosis ($P < 0.001$) with the ninth thoracic vertebra as the mode. Computerised axial tomography demonstrated constant findings (Fig. 4). While the apical vertebrae had spun furthest from the neutral position they were least rotated one to another. Maximum intervertebral rotation occurred at the junctions of the structural curve and its upper and lower compensatory curves; this was strictly in the nature of derotation bringing the spine neutral above and below.

Cadaveric analysis. The appearance of the specimens from the front and side demonstrated a mean Cobb angle for the scoliosis of 67 degrees and a mean overall angle of kyphosis of 76 degrees. This apparent kyphosis was entirely spurious, the scoliosis itself being seen in two planes at right angles, which are neither anteroposterior nor lateral projections of the apex of the curve because in severe cases the spine is directed not only sideways but backwards in the region of the scoliosis (Figs 5 to 10).

When true anteroposterior and lateral projections of the apex were measured, the mean Cobb angle of scoliosis was increased to 90 degrees, and there was a mean lordosis at the apex measuring three degrees. Computerised axial tomography showed rotational changes similar to those of the patients. Computerised vertical tomography demonstrated that it was possible to obtain the entire specimen on one longitudinal slice measuring 12 mm, confirming that a kyphosis did not exist (Fig. 11).

Biomechanical studies. Figure 1 shows the effect of flexion of a lordotic segment of the spine with pre-existing coronal plane asymmetry. The force of forward flexion (F) produces tightening of the posterior structures (T) and a reactive force (f). The horizontal component of f acting at a distance (d) from the midline will produce a spinning moment (M) with the spinous processes being drawn back to the midline. The magnitude of this spinning moment is therefore a function of the force of forward flexion and degree of biplanar asymmetry. Asymmetric vertebral shape in the transverse plane

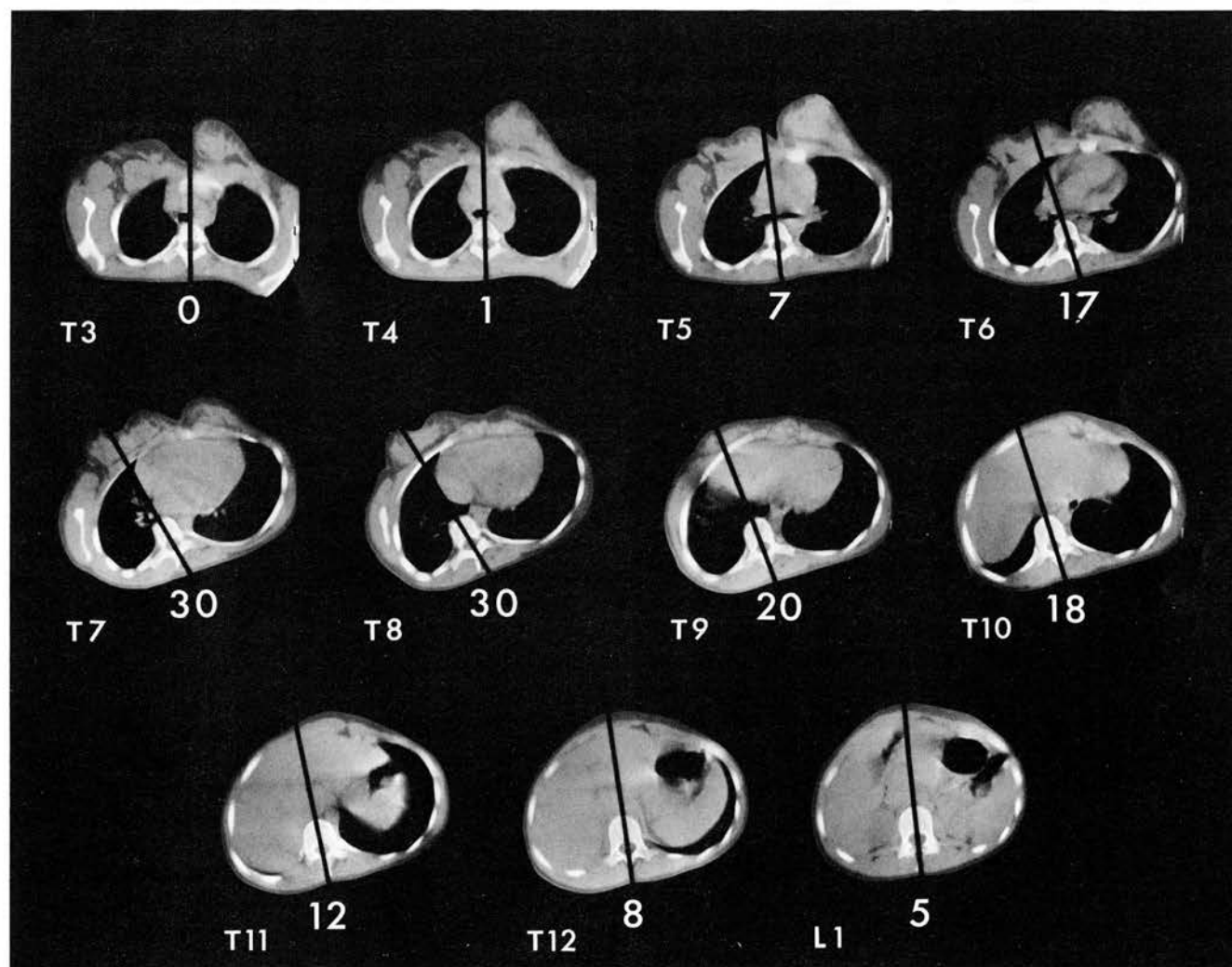


Fig. 4

Computerised axial tomograms of an idiopathic right thoracic curve. Sections have been taken transversely through each vertebra in the curve from T3 to L1 with the angle of rotation measured at each level. The apical vertebrae, T7 and T8, are not rotated one to another.

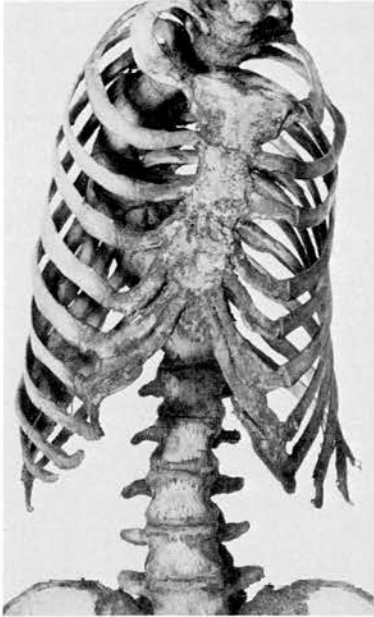


Fig. 5



Fig. 6



Fig. 7



Fig. 8



Fig. 9



Fig. 10

Figures 5 and 6—Anteroposterior photograph and radiograph of a specimen with severe right thoracic scoliosis. Figures 7 and 8—Lateral photograph and radiograph of the same specimen showing an apparent and spurious kyphosis. Figures 9 and 10—True lateral photograph and radiograph of the apex of the curve demonstrating a lordosis.

would produce the same effect as coronal plane asymmetry. Median plane asymmetry does not have to be in the nature of a lordosis, as flat or slightly kyphotic segment models satisfy the rotational requirements.

Forward flexion of the rabbit thoracolumbar spine to the limits of normal movement is not coupled with any axial rotation. When specimens with biplanar asymmetry are flexed, then the relative tightening of the posterior structures (or excessive length of the anterior structures) creates a tendency for the spine to rotate, precipitated by any inherent coronal plane asymmetry in the system (Figs 12 and 13). The effect of rotation on this lordotic region is such that the vertebral bodies lie on the convexity and the posterior elements on the concavity. Excessive forward flexion of the normal spine beyond the limits of compression of the anterior structures does produce rotation but with the bodies directed to the concavity and the posterior elements to the convexity as would be expected with a rotated kyphosis.

DISCUSSION

The anatomical planes of the body are the coronal plane (dividing the body into front and back portions), the median (sagittal) plane (dividing the body into right and left halves) and the transverse or horizontal plane (any cross-section) (Brash 1951). In the median plane the spine has four symmetrical curvatures, cervical and lumbar lordoses and thoracic and sacral kyphoses. The

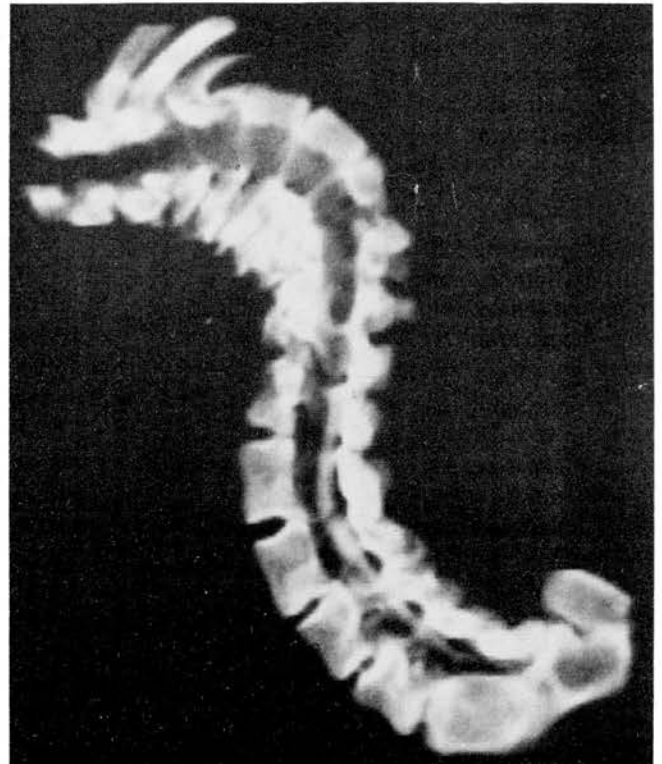


Fig. 11

Computerised vertical tomography of a cadaveric specimen with severe idiopathic scoliosis. The entire specimen is obtainable in one longitudinal slice. A kyphosis cannot exist.

deformity of idiopathic scoliosis appears as a curvature in the coronal plane but this deformity is entirely secondary. It is the product of lordosis plus rotation as Adams stated in 1865. Indeed this must be the case as rotation always occurs in the same direction, with the spinous processes directed towards the concavity, whereas in a rotated kyphosis the spinous processes are always directed towards the convexity (Somerville 1952).

It is a constant clinical feature in idiopathic scoliosis that the normal spinal curvatures in the median plane are altered—reduced in the thoracic region with thoracic curves. Willner (1981) measured the overall thoracic kyphosis and demonstrated statistically that patients with idiopathic thoracic scoliosis had flatter thoracic kyphoses than normal children, whose kyphoses in turn were flatter than patients with Scheuermann's disease. However, his measurements were made from the top to the bottom of the thoracic kyphosis and he did not look at the median plane in the region of the apex of the curve.

When this is scrutinised there is always evidence of a lordosis or lordotic tendency and this is clearly visible from modest scolioses to the severest specimens in pathology museums. Furthermore, it is this appearance which differentiates progressive from non-progressive scolioses detected by school screening. When true lateral projections of the apex are obtained, there is clear evidence of asymmetric vertebral growth, with anterior body height greater than posterior. The findings of computerised axial tomography indicate that it is the area of lordosis as a whole that has spun out from the neutral plane, there being much less intervertebral rotation occurring at the apex.

Importantly, spinal curvatures in the median plane change during growth and in normal children the thoracic kyphosis reduces in size from the age of 8 to 14 years, reaching a minimum at about the age of 12. That this occurs at the same time in both boys and girls demonstrates its independence from growth velocity,

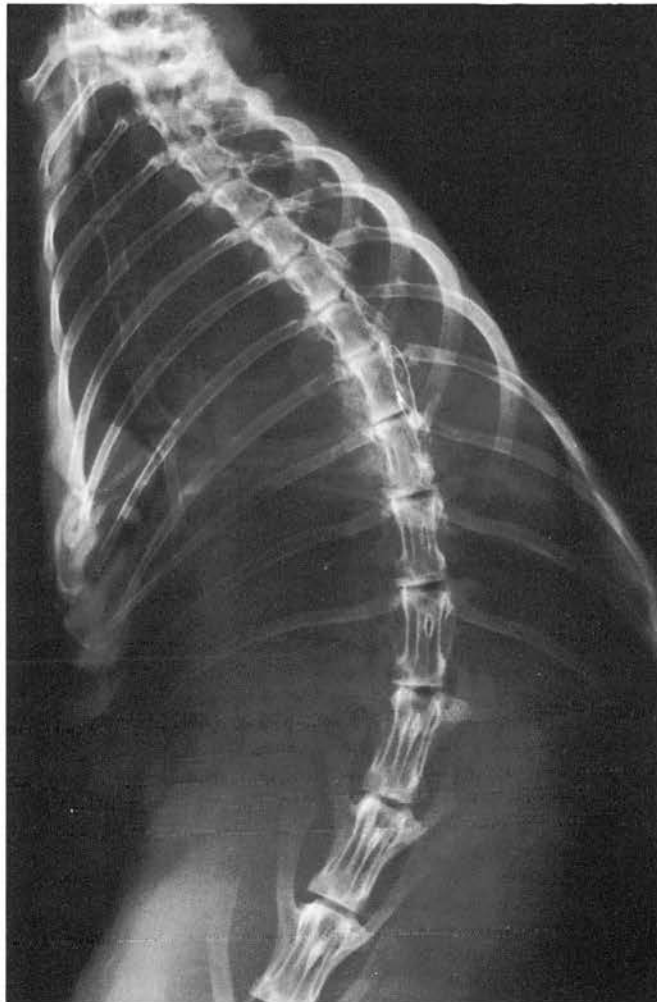


Fig. 12

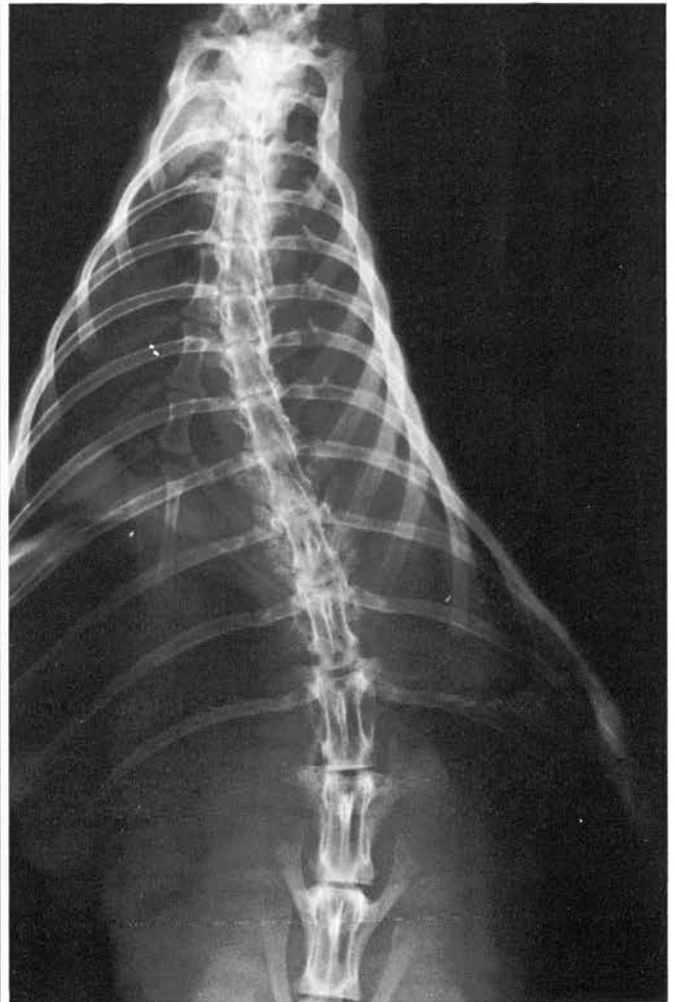


Fig. 13

Figure 12—Anteroposterior view of a fresh normal rabbit spine that has been flexed beyond the normal limits. A lateral curvature of the spine has been produced but the thoracic spinous processes are directed towards the convexity as would be expected with a rotated kyphosis. Figure 13—Anteroposterior view of a fresh rabbit spine with thoracic biplanar asymmetry which has been similarly flexed. A thoracic scoliosis has been produced but the spinous processes are directed towards the concavity.

boys on average maturing some two years later. However, when the thoracic kyphosis is at its minimum in girls, they are going through peak adolescent growth velocity and this may explain greater progression potential in girls. When the thoracic kyphosis is at its minimum in boys their growth velocity is constant but when their thoracic kyphosis becomes maximal in later adolescence they are then going through peak adolescent growth velocity, which may explain why boys are particularly prone to developing Scheuermann's disease, the opposite median plane deformity.

Interestingly, it is during this phase of reduction in thoracic kyphosis that the incidence of scolioses detected in the community rises sharply in both boys and girls (Dickson 1983). The cadaveric analysis reinforces the need to think and see in three dimensions. Idiopathic kyphoscoliosis does not exist at any magnitude even in the most severe specimens. The spurious appearance of a kyphosis when the patient is viewed from the side is the scoliosis seen in a plane which is more an anteroposterior projection of the apex of the curve than an AP projection of the patient or specimen, compounded to a lesser extent by the deceptive rib hump. If the whole of the spinal canal can be visualised from upper cervical to lower lumbar region in one longitudinal slice, then a kyphosis cannot possibly exist.

The apical site in thoracic idiopathic scoliosis is bimodal, the great majority being apical at the T8-9 level with a small minority apical at the T3 level. These are important regions of the spine in both median and transverse planes. Anatomists have recorded in detail that while the thoracic spine extends from T1 to T12 by convention this is not anatomically so (Inkster 1951). The first two and last three thoracic vertebrae resemble typical cervical and lumbar vertebrae respectively. Typical thoracic vertebrae only extend from T3 to T9. The potential rotational instability of the cervical and lumbar lordoses is counteracted by the shape of these vertebrae in the transverse plane. The increased lateral to anteroposterior width (a prism with its base anteriorly) confers great rotational stability aided by the powerful posterior muscular, ligamentous and fascial support in the cervical and lumbar regions.

In contradistinction the heart-shaped thoracic vertebra (a prism with its apex anteriorly) is rotationally unstable and is therefore protected by the presence of a kyphosis, which places the axis of spinal rotation anterior to the vulnerably shaped bodies. In the true lateral projections of patients and specimens there was no compensatory or associated kyphosis below the area of lordosis, which was therefore a lumbar lordosis extending abnormally high. At the apex of the curve there is now a vertebra heart-shaped in the transverse plane, and computerised axial tomography confirms this, which is not protected by a kyphosis. Only a mild lordosis or lordotic tendency is sufficient to cause the spine to rotate to the side and, once the apex has rotated, the posterior

part of the end-plate is relieved from direct compression. This is verified by the observation that the size of the lordosis does not correlate with the size of the scoliosis. The negative correlation with the angle of the total kyphosis shows that it is the size of the lordosis which relates to the amount of reduction of the overall kyphosis.

The concept of a short-segment lordosis being an important factor has only received sporadic support (Heuer 1927; Somerville 1952; Roaf 1966; Deane and Duthie 1973). Figure 13 clearly shows the spinning potential of a lordosis but the system would be in equilibrium even in extreme forward flexion unless there was spinal asymmetry in another plane. Biplanar asymmetry is therefore the crucial factor and such a spine will readily deform accordingly. Somerville was a pioneer in this respect and produced a progressive "idiopathic" scoliosis in three rabbits by causing a short-segment lordosis. He must, however, have inadvertently caused a few degrees of coronal plane asymmetry, as when this work was repeated by others (Wittebol 1956; Gottlieb 1957) the spine remained disappointingly straight. This is because animals, unlike so many normal children detected in the community by school screening programmes, have no recorded tendency to have a pre-existing coronal plane curvature which provides rotational instability. "Idiopathic" scoliosis can only be produced in animals if asymmetry in two planes is produced at the same time (Dickson *et al.* 1983). The younger the patient or animal, the more serious is the progression potential because of the overall environment of growth. While the epiphyses of the long bones and pelvis are fused by the eighteenth year, the vertebral epiphyses do not do so until the twenty-fifth (Inkster 1951) and this may explain the recent finding that the majority of idiopathic curves progress until that time (Hassan and Bjerkreim 1983).

Normal spinal asymmetry also exists in the transverse plane, particularly in the thoracic region, where the thoracic vertebrae from T5 to T8 are constantly grooved on the left side by the pulsations of the descending aorta, and this is more obvious when the thoracic kyphosis is reduced, bringing spine and artery in apposition. The thoracic vertebrae are therefore asymmetric prisms and the presence of a lordosis without coronal plane asymmetry will produce rotation with the convexity to the right. The primary asymmetry is in the body, pedicle asymmetry being secondary to the scoliosis (Karaharju 1967). Indeed, a minor degree of transverse plane asymmetry may be the factor producing so much coronal plane asymmetry observed in normal children. This is reinforced by the observation that many normal children (children without a scoliosis) have a slight rib hump on the right side on forward flexion (Burwell *et al.* 1982). This of course is asymmetric buckling of a normal thoracic kyphosis but when there is a lordotic region in the lower thoracic spine a true rotational lordosis produces a progressive idiopathic curve.

It is therefore the lateral profile of the spine which is the key to the understanding of progressive idiopathic deformities. Both idiopathic scoliosis and Scheuermann's disease have been shown to have a not dissimilar incidence and a familial trend (Sørensen 1964; Wynne-Davies 1965). As virtually all aspects of growth are governed by genetic mechanisms it may well be the lateral profile which is genetically determined. At the extremes of the spectrum of lateral profile are on the one hand those with the most pronounced median plane curvatures in whom Scheuermann's disease is the clinical manifestation, and on the other those with the flattest profile who are particularly vulnerable to developing a rotational lordosis. The flat lateral profile of patients with idiopathic scoliosis also explains the observation that these children are taller than their peers but not growing faster (Dickson and Sevitt 1982). This is more likely the effect of the lateral profile than genetic tall stature as the condition affects children of all sizes.

This concept of biplanar spinal asymmetry more than adequately explains the clinical behaviour of idiopathic scoliosis. On forward flexion the rotational prominence increases, being maximal on full flexion. The greater the flexion, the less the relatively increased anterior vertebral height at the apex can be accommodated without rotating to the side. Non-structural scolioses barely rotate because their asymmetry does not affect the lateral profile. Conservative treatment, be it brace or cast, is effective primarily in preventing spinal flexion but cannot, however, be corrective as only the *status quo* of the secondary deformity is preserved while the primary lordosis may in fact be increased. That brace-wearing is only at best holding and is never corrective testifies to this mode of action, but as the curve increases, so the brace is less effective as a holding device because the increasingly adverse effects of gravity cannot be attenuated merely by preventing flexion.

Underarm orthoses are effective if the apex of the curve is not above the eighth thoracic vertebra because higher curves can still flex. A crucial part of brace design

is that the lumbar lordosis should be obliterated (Blount and Moe 1973) and this produces the well-recognised phenomenon of a temporary partial correction while in the brace. Obliteration of the lumbar lordosis produces compensatory thoracic hyperextension, providing more room for the thoracic vertebral bodies, thus causing a derotation effect. While animal models of idiopathic scoliosis have tended to resist development until recently, there has always been one superb human model. It is critical when fusing a spine for idiopathic scoliosis to do so from neutral vertebra above to neutral vertebra below (Goldstein 1973). Too short a fusion is the most common cause of deterioration of the curve after the operation, sometimes at a greater rate than before (Roaf 1966). This is because the primary lordosis has been augmented by too short a fusion, which acts as a tethering strut facilitating rotational progression.

Current methods of treatment for idiopathic scoliosis are directed entirely towards the secondary deformity. While bracing may at best prevent progression, surgical procedures at best improve the coronal plane curvature component by 50 per cent, with a much less satisfactory correction of the rotational prominence. Elucidation of the pathogenesis of idiopathic scoliosis enables treatment to be directed towards the primary deforming mechanisms, of which the lateral profile is the more important. If the normal thoracic kyphosis is recreated at the apex of the curve in the experimental animal, then the secondary deformity resolves with growth (Dickson *et al.* 1983). This can be achieved clinically by lifting the posterior elements of the apical vertebrae back to a normal kyphosis. Without spinal fusion the spine may grow straight and thus biological treatment can be considered.

As with many orthopaedic conditions by the time they achieve clinical significance, it is the secondary deformity which is so obvious and masks or obscures the underlying primary deformity. The lateral curvature of the spine with rotation is secondary. Biplanar spinal asymmetry during growth is the essential lesion.

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REFERENCES

- Adams W. *Lectures on the pathology and treatment of lateral and other forms of curvature of the spine*. London: Churchill and Sons, 1865.
- Alexander MA, Season EH. Idiopathic scoliosis: an electromyographic study. *Arch Phys Med Rehabil* 1978; **59**: 314-5.
- Boas F. Studies in growth. *Hum Biol* 1932; **4**: 307-50.
- Blount WP, Moe JH. *The Milwaukee brace*. Baltimore: Williams & Wilkins, 1973.
- Brash JC. *Cunningham's text-book of anatomy*. 9th ed. London: Oxford University Press, 1951: 3-4.
- Burwell RG, James NJ, Johnson F, Webb JK. The rib hump score: a guide to referral and prognosis? *J Bone Joint Surg [Br]* 1982; **64-B**: 248.
- Deane G, Duthie RB. A new projectional look at articulated scoliotic spines. *Acta Orthop Scand* 1973; **44**: 351-65.
- Dickson RA. Scoliosis in the community. *Br Med J* 1983; **286**: 615-8.
- Dickson RA, Lawton JO, Archer IA, *et al.* Combined median and coronal plane asymmetry: the essential lesion of progressive idiopathic scoliosis. *J Bone Joint Surg [Br]* 1983; **65-B**: 368.
- Dickson RA, Sevitt EA. Growth and idiopathic scoliosis: a longitudinal cohort study. *J Bone Joint Surg [Br]* 1982; **64-B**: 385.
- Goldstein LA. The surgical treatment of idiopathic scoliosis. *Clin Orthop* 1973; **93**: 131-57.

- Gottlieb O. Experimental scolioses (advance report). *Acta Orthop Scand* 1957; **26**: 295-6.
- Hassan I, Bjerkreim I. Progression in idiopathic scoliosis after conservative treatment. *Acta Orthop Scand* 1983; **54**: 88-90.
- Heuer F. Zur Theorie der Scoliose. *Hessisches Ärzteblatt* 1927 No. 18 and ff. (Cited by Wittebol P, Idiopathic scoliosis (an experimental investigation). *Arch Chir Neerl* 1956; **8**: 269-79.)
- Inkster RG. Osteology. In: Brash JC, ed. *Cunningham's text-book of anatomy*. 9th ed. London: Oxford University Press, 1951: 105.
- Kaneko T. Histopathological and histochemical studies on the back muscles in scoliosis. *J Japan Orthop Assoc* 1968; **42**: 13-28 (in Japanese).
- Karaharju EO. Deformation of vertebrae in experimental scoliosis: the course of bone adaptation and modelling in scoliosis with reference to the normal growth of the vertebra. *Acta Orthop Scand* 1967; Suppl 105.
- Khosla S, Tredwell SJ, Day B, Shinn SL, O'valle WK Jr. An ultrastructural study of multifidus muscle in progressive idiopathic scoliosis: changes resulting from a sarcolemmal defect at the myotendinous junction. *J Neurol Sci* 1980; **46**: 13-31.
- Lerique and Le Coeur. Action potential asymmetry on two sides. 1951. (Cited by Le Febvre J, Triboulet-Chassevant A, Missirlu MF. Electromyographic data in idiopathic scoliosis. *Arch Phys Med Rehabil* 1961; **42**: 710-1.)
- Roaf R. The basic anatomy of scoliosis. *J Bone Joint Surg [Br]* 1966; **48-B**: 786-92.
- Sabatier. *Traité complete d'anatomie*. Paris, 1777. (Cited by Farkas A. Physiological scoliosis. *J Bone Joint Surg* 1941; **23**: 607-27.)
- Sahlstrand T, Petruson B. A study of labyrinthine function in patients with adolescent idiopathic scoliosis. I. An electro-nystagmographic study. *Acta Orthop Scand* 1979; **50**: 759-69.
- Somerville EW. Rotational lordosis: the development of the single curve. *J Bone Joint Surg [Br]* 1952; **34-B**: 421-7.
- Sorensen ICH. *Scheuermann's juvenile kyphosis*. Copenhagen: Munksgaard, 1964.
- Sørensen K. *Scheuermann's juvenile kyphosis: clinical appearances, radiography, aetiology and prognosis*. Copenhagen: Munksgaard, 1964.
- Stagnara P, Fleury D, Fauchet R, et al. Scolioses majeures de l'adulte supérieures à 100 degrés: 183 cas traités chirurgicalement. *Rev Chir Orthop* 1975; **61**: 101-22.
- Staub HA. Eine skoliotikerfamilie. Ein beitrag zur frage der kongenitalen skoliose and der hereditat der skoliosen. *Z Orthop Chir* 1922; **43**: 1-20.
- Trontelj JV, Pečak F, Dimitrijević MR. Segmental neurophysiological mechanisms in scoliosis. *J Bone Joint Surg [Br]* 1979; **61-B**: 310-3.
- Whittle MW, Evans M. Instrument for measuring the Cobb angle in scoliosis. *Lancet* 1979; **1**: 414.
- Willner S. Spinal pantograph—a non-invasive technique for describing kyphosis and lordosis in the thoraco-lumbar spine. *Acta Orthop Scand* 1981; **52**: 525-9.
- Wittebol P. Idiopathic scoliosis (an experimental investigation). *Arch Chir Neerl* 1956; **8**: 269-79.
- Wynne-Davies R. Familial scoliosis. In Zorab PA, ed. *Proceedings of a symposium on scoliosis: held in the Institute of Diseases of the Chest, The Brompton Hospital, London on 5 and 6 July 1965*. Action for the Crippled Child Monograph. London: National Fund for Research into Poliomyelitis and other Crippling Diseases, 1965: 36-41.
- Yamada K, Takaaki I, Yamamoto H, Nakagawa Y, Tanaka H, Tezuka A. Equilibrium function in scoliosis and active corrective plaster jacket for the treatment. *Tokushima J Exp Med* 1969; **16**: 1-7.

THE PATHOGENESIS OF IDIOPATHIC SCOLIOSIS.

Dickson RA, Lawton JO, Butt WP.

In 'Management of Spinal Deformities' (Dickson RA and
Bradford DS Eds) Butterworth International Medical Reviews:
London etc, 1-37, 1984.

This textbook chapter expanded the concepts put forward in the previous paper (Reference 11) and introduced observations and data explaining the laterality of spinal curvatures (a preponderance for right sided curves in the thoracic region and left in the lumbar region) and the different prevalence rates in boys and girls (the former being relatively protected from progression and the latter being more vulnerable). This chapter also introduced the concept that idiopathic scoliosis was the opposite condition to Scheuermann's disease (idiopathic kyphosis) and put forward a more physiological approach to the surgical treatment of idiopathic thoracic deformities principally addressed to the sagittal profile.

1

The pathogenesis of idiopathic scoliosis

Robert A. Dickson, J. O. Lawton and W. P. Butt

INTRODUCTION

Orthopaedic surgeons are frustrated in their attempts at treating many musculo-skeletal deformities in childhood because by the time the condition has achieved clinical significance it is compounded by secondary deformities which mask or even totally obscure the underlying primary lesion. The temporary period of perinatal hip joint laxity producing a congenital dislocation of the hip is a notable example. Unless detected at birth, and sometimes even if so, the secondary deformities of bone, joint and soft tissue completely preclude the possibility of restoration to a normal situation because these have become autonomous in their own right. If the secondary deformities are particularly marked, then the best that treatment can achieve is to reduce the deformity as far as possible and prevent it from recurring during subsequent growth.

In this respect idiopathic scoliosis is no exception. Clinical manifestations of the curvature become apparent when secondary deformities have already arisen (*Figure 1.1*), as evidenced by the fact that the curve magnitude at presentation in the UK is still of the order of 50° (Belstead and Edgar, 1978). Hence treatment has been directed towards the secondary deformities, either holding the curve with a brace or achieving the best possible correction of the lateral curvature by spinal instrumentation and preventing further progression by spinal fusion. The inadequacy of such treatment is self-evident from the fact that the optimum result of conservative treatment is merely to prevent the curve from deteriorating further and that operative treatment rarely offers a better than 50% correction of the lateral curvature, often with much less effect on the vertebral rotation. However, by an understanding of the pathogenesis of scoliosis it is possible to direct treatment towards correcting the essential lesion. As a result, subsequent growth is no longer a factor tending to mitigate the effects of treatment but becomes a powerful positive force acting to correct the secondary deformities. How, then, does a spine



Figure 1.1 Anteroposterior radiograph of a right thoracic idiopathic scoliosis; there is a lateral curvature with rotation and rib asymmetry (For a better quality picture see Publication 25, Figure 1.)

which apparently started straight bend and twist out to the side? Although there is no clinical evidence of either nerve or muscle dysfunction in idiopathic scoliosis, most workers favour a neuromuscular basis for this condition, with growth and genetic considerations also thought to be important.

NEUROMUSCULAR FINDINGS

After Lerique and Le Coeur in 1951 demonstrated action potential differences on the two sides of a scoliotic spine, muscle imbalance was put forward as a possible cause of idiopathic scoliosis (Riddle and Roaf, 1955).

Electromyography

Early promising electromyographic evidence of increased activity on the convex side (Weiss, Milkowska and Kozinska, 1957) was, however, found in non-idiopathic patients. Subsequent reports of increased activity on the convex side in prone patients with idiopathic scoliosis (Le Febvre, Triboulet-Chassevant and Missirliu, 1961; Henssge, 1964) were invalidated by improper patient positioning (Alexander and Season, 1978). The findings of fibrillation potentials indicative of denervation in more than 50% of patients with idiopathic scoliosis (Henssge, 1968; Peretti and Velluti, 1969) were negated by Alexander and Season's careful work which showed that these denervation potentials were in fact nothing more than endplate noise and they agreed with Zuk (1962) who first demonstrated that these electrical abnormalities were attributable to the presence of a spinal curvature. That these electrical changes are secondary is supported by the observations that the spine becomes silent after fusion even though the curve remains, that activity returns in the presence of a pseudarthrosis (Butterworth and James, 1969), and that activity tends to become more silent in the Milwaukee brace (Alexander and Season, 1978). Furthermore, it has been clearly shown that in normal children without scoliosis asymmetric motor activity can be induced merely by causing a non-structural lateral curvature of the spine by positioning (Goodgold, 1974). More recently Trontelj, Pecak and Dimitrijevic (1979) have demonstrated different activities in the superficial and deep muscle layers of the back but could find no evidence of a primary deforming mechanism.

Structure and ultrastructure

Undeterred by the lack of electrical evidence, workers scrutinized the muscles of the back. Gorynski and Bojkowa (1957) demonstrated histological abnormalities of the spinal muscles in dystonic scoliosis but James, Lloyd-Roberts and Pilcher (1959) could find no such changes in infants with idiopathic scoliosis. Kaneko in 1968 did demonstrate histopathological and histochemical abnormalities of the spinal muscles in idiopathic scoliosis and an electronmicroscopic study in 1972 by Hirano showed signs of both dystrophy and atrophy of the back muscles. Fidler, Jowett and Troup (1974) demonstrated histochemically a greater proportion of slow twitch versus fast twitch fibres at the apex on the convex side and, with their studies of the length of the multifidus muscle (Fidler and Jowett, 1976), assumed a primary muscle imbalance mechanism for idiopathic scoliosis. Spencer and Zorab (1976), while confirming the presence of abnormalities, could find no particular site nor side. Spencer and Eccles (1976) found more type II fibres at the apex in the concavity in patients with idiopathic scoliosis, but no such changes were found when non-structural scoliosis was cast-induced in rabbits. Ultrastructural changes were also observed in southern Chinese adolescents with idiopathic scoliosis (Wong *et al.*, 1977) but there were no differences found either between sides or particular muscle groups. Yarom and Robin (1979) suggested a generalized neuromuscular

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disorder on the basis of mild type I atrophy on the concave side and in the deltoid muscles.

The correct interpretation of these investigations is made all the more difficult as idiopathic scoliosis is a three-dimensional deformity (*Figure 1.2*) with as much rotation as lateral deformation and if the deformity is of the magnitude that requires surgical correction (the biopsies for these investigations were taken during corrective surgery) then it is too simple to consider the paraspinal musculature to be either on the convex or concave sides of the curve. With significant rotation the concave paraspinal musculature actually lies anteriorly. More importantly, it is not possible to exclude an iatrogenic cause for these structural and ultrastructural

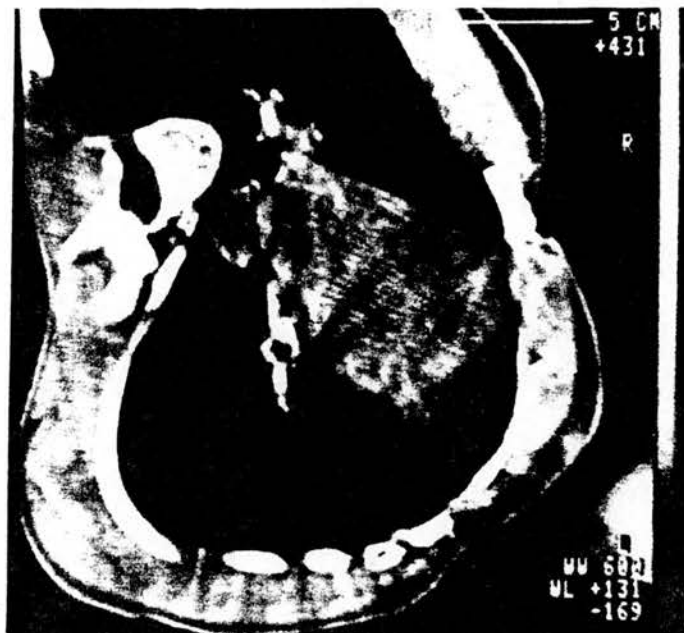


Figure 1.2 Computerized axial tomograph of the apex of a 45° idiopathic curve: there are 45° of apical rotation

changes suggestive of neuropathy, myopathy, fibrosis and frank neuromuscular damage. Some form of preoperative traction was frequently used prior to surgical correction with the specific objective of disrupting muscle or ligament by tension and it is well established that such treatment can disrupt both the central and peripheral nervous systems (MacEwen, Bunnell and Sriram, 1975). The most recent ultrastructural study would entirely support this, damage and fibrosis being found on the concave side (Khosla *et al.*, 1980). Idiopathic scoliosis commences with a straight spine in the coronal plane (*Figure 1.3*), and, therefore, to be meaningful structural and ultrastructural studies must be performed at an earlier stage before shape has been influenced by treatment, growth, or the presence of

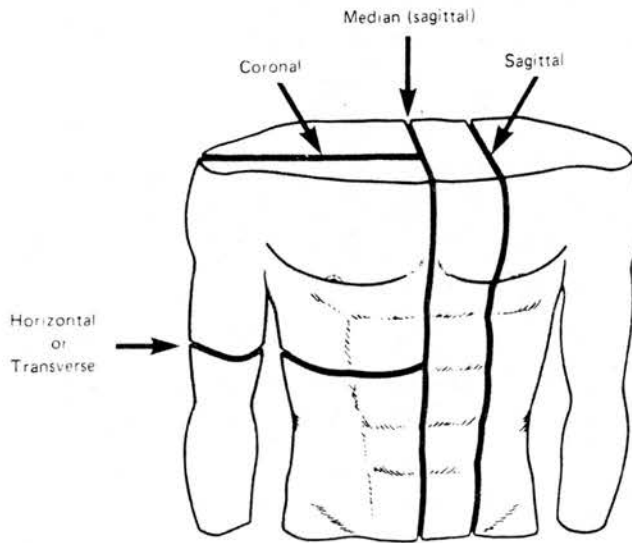


Figure 1.3 The anatomical planes of the body

the secondary deformity. The finding of virus-like particles of a glycogenic nature in the paraspinal muscles in four patients with scoliosis (Webb and Gillespie, 1976) is misleading. One of the four patients had a congenital scoliosis and in any event such particles are not uncommon findings in normal muscle (Caulfield, Rebeiz and Adams, 1968), as any experienced neuropathologist will readily confirm.

Equilibril function

In the search for some evidence in support of a neuromuscular basis equilibril function was investigated and abnormalities were noted to be more prevalent in patients with idiopathic scoliosis than controls (Yamada *et al.*, 1969). An increased prevalence rate of scoliosis in children with retarded development of equilibril function was also observed (Yamada and Yamamoto, 1972). Increased postural sway during labyrinthine stimulation on the concave side suggested either asymmetric sensitivity in the labyrinth or dysfunction of postural control mechanisms at brainstem level (Sahlstrand, Petruson and Ortengren, 1979). When this was investigated electronystagmographically an increased occurrence of spontaneous and positional nystagmus was noted in patients with adolescent idiopathic scoliosis; no differences were observed with respect to curve size (Sahlstrand and Petruson, 1979a) and it was conceded that the observations could be caused by feedback from a deformed spine. No electronystagmographic differences were observed between the erect and supine positions (Sahlstrand and Petruson, 1979b), which was interpreted as more in favour of a primary mechanism, although the importance of curve rigidity was not investigated.

GROWTH AND GENETIC THEORIES

There is more of substance to growth and genetic studies. Growth is clearly an important environment in which idiopathic scoliosis not only commences but may progress until maturity and often after (Leatherman and Dickson, 1979; Hassan and Bjerkreim, 1983). It is growth as a background factor rather than peak growth velocity which is important (Duthie, 1959). Although Duval-Beaupere in 1979 could not observe any abnormal growth pattern in scoliosis patients, it was subsequently suggested that girls with adolescent idiopathic scoliosis were taller than their straightbacked counterparts, being more advanced early in adolescence and more retarded later (Willner, 1974; Nordwall and Willner, 1975). That increased stature could be important in the development and progression of idiopathic scoliosis received further support from an investigation of the development of southern Chinese girls with adolescent idiopathic scoliosis (Low *et al.*, 1978). However, these studies compared the standing height of girls with adolescent idiopathic scoliosis with norms obtained at least a generation earlier and are therefore unsound as normal children will appear significantly taller than norms obtained 20 years earlier. Indeed, one study (Willner, 1974) demonstrated that female controls grew over 2 cm from the age of 16 to 18 years. Such a growth pattern is quite abnormal (Tanner, Whitehouse and Takaishi, 1966). A recent prospective study using contemporaneous controls demonstrated no differences in growth velocity or development (Dickson and Sevvitt, 1982) (*Figure 1.4*). A

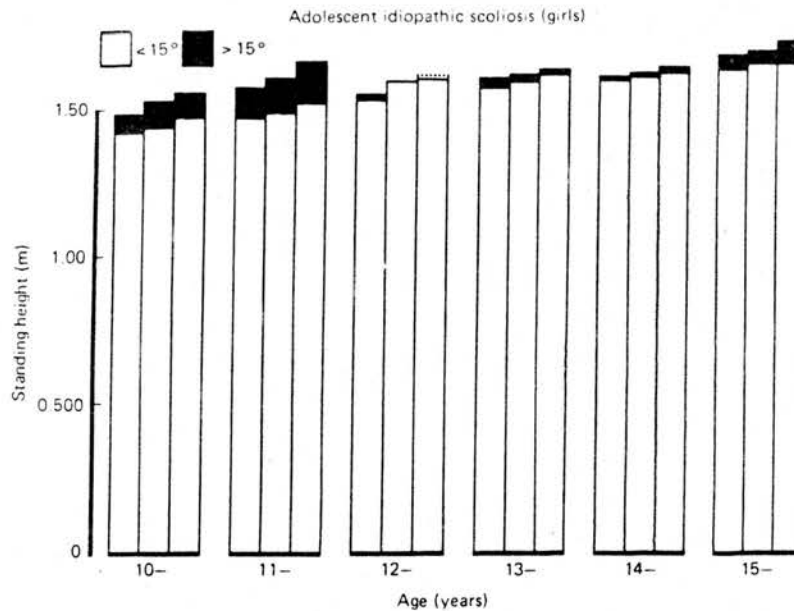


Figure 1.4 Histogram of standing height of girls aged 10–15. The three columns each year represent heights, initially, at 6 months and at 1 year. Shaded areas indicate the increased height at each age of girls with progressive idiopathic scoliosis, with the exception of the 1-year follow-up of 12-year-olds

significant trend was, however, observed for girls with progressive curves to be taller than their counterparts with small and static curves, suggesting that stature, not necessarily growth, is very relevant.

Since scoliosis was first described in twins (Faber, 1935) and families (Staub, 1922) much interest has been focused on the genetic aspects of idiopathic scoliosis. In 1936 Faber demonstrated that while congenital scoliosis was not inherited, rachitic scoliosis was. There is no doubt that many of these individuals must have had idiopathic scoliosis. A resurgence of interest in the genetic aspects of idiopathic scoliosis followed the work of Wynne-Davies who demonstrated in 1965 that there was a much increased prevalence of idiopathic scoliosis in relatives of patients, particularly of the first degree. The familial nature of this condition has since been confirmed (DeGeorge and Fisher, 1967; Cowell, Hall and MacEwen, 1972;

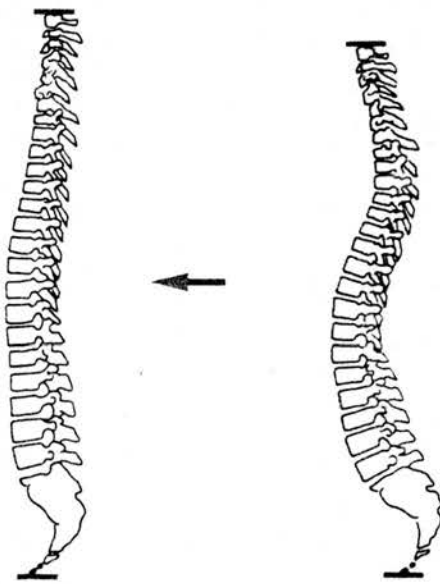


Figure 1.5 Standing height is increased when the normal curvatures in the median plane are suppressed, producing an uncoiling effect

Riseborough and Wynne-Davies, 1973; Robin and Cohen, 1975; Czeizel *et al.*, 1978). Wynne-Davies, could not, however, determine whether the condition was of dominant or multiple gene inheritance while Cowell, Hall and MacEwen suggested both dominant and sex-linked patterns but with variable expressivity and incomplete penetrance. Comparisons between these studies are not facilitated by the use of different thresholds for the diagnosis of a scoliosis. While Riseborough and Wynne-Davies in Boston used 20° , Cowell, Hall and MacEwen used 10° , and the latter study must therefore have been significantly diluted by large numbers who did not have idiopathic curves.

Adolescence is, however, the only period during growth since 4 months of fetal age that growth velocity actually increases and the fundamental control of development is clearly genetic (Tanner, 1962). The correspondence between mothers and sisters, between sisters, dizygotic twins, and identical twins in relation

to time of menarche, peak height growth velocity, and skeletal maturity, demonstrates this clearly (Boas, 1932; Bolk, 1923; Hewitt, 1957; Sontag and Nelson, 1933). Indeed, parental height still remains the most accurate single occasion guide to the ultimate height a child will attain (Tanner, 1962). That girls with idiopathic scoliosis of bigger curve magnitude are taller than girls with smaller curves, have always been tall, and are not growing faster, suggests that these children have genetic tall stature. There will therefore be a trend for their families to be tall, in whom one would expect to find a higher prevalence of idiopathic scoliosis if the condition were in that way growth-related. Clearly it would be unwise for the trend to rename idiopathic scoliosis 'familial' or 'genetic scoliosis' to continue and such a caution has already been issued (Editorial, *British Medical Journal*, 1975). However, the increased stature is better explained by suppression of normal spinal curves in the median plane (*Figure 1.5*), which relatively uncoils the individual with the progressive scoliosis (Dickson *et al.*, 1983).

EXPERIMENTAL WORK

Attempts to create a scoliotic deformity in the experimental animal have concentrated on procedures affecting either the stability or the growth of the spine. Schwartzmann and Miles (1945) performed an extensive series of operations on rats and mice, excising or releasing deep and superficial musculature. On each occasion they produced a paralytic type of curve which was convex to the side of the operation. Other workers looked at the effect of muscle denervation. Bisgard (1935) divided the intercostal nerves in rabbits and goats and again produced a paralytic type of curve but found that with follow-up the curve rapidly corrected. In a more comprehensive study of the effects of nerve division Liszka (1961) studied the effects of division of both anterior and posterior spinal roots in the lower thoracic region of rabbits. He was able to create a curvature by either method and inferred that interruption of the normal reflex arc was an important factor. His work was repeated by MacEwen (1968) with similar results. However, when Alexander and Season (1978) performed a similar study they found that whereas in all animals where anterior and posterior roots had been divided a lateral curvature developed, this only occurred in 60% of animals where the posterior roots alone had been divided. When they examined the spinal cord of these animals microscopically they found that in those animals where posterior root section alone had produced a curvature there was evidence of anterior horn chromatolysis, whereas this was not present in the 40% which showed no curvature. They concluded, therefore, that the final common pathway remained the efferent supply to the muscles producing a paralytic type of deformity.

An exhaustive set of experiments was carried out in rabbits and pigs by Langenskiöld and Michelsson (1961), variously dividing muscles, ligaments and ribs in an attempt to produce a scoliotic deformity. The most consistent results followed the excision of the neck and head of the ribs, creating an immediate functional curve convex to the side of the operation. Most of these curves subsequently progressed with time, although some in fact regressed. They also demonstrated that a lateral curvature of the spine followed hemilaminectomy over

several levels in about two-thirds of animals. They felt that the common factor was division of the posterior costotransverse ligament, although admitting that a severe progressive curvature only developed in a few animals where this ligament was sectioned alone. This was reaffirmed by Manning (1968), who, attempting to repeat their work, found that division of the costotransverse ligament alone was unsuccessful whereas excision of the heads of the ribs produced a spinal deformity. A similar outcome was achieved by Piggott (1971), again using rabbits. However, following the excision of rib heads in primates by Robin and Stein (1975) the spine remained frustratingly straight.

The mature scoliotic spine exhibits both lateral deviation with wedging of the vertebrae in the coronal plane and rotation of the vertebrae associated with asymmetry of pedicle length. This had led workers to try to recreate these essentially secondary deformities by interfering with vertebral growth. Unilateral trauma to the vertebral growth plate by Haas (1939) using dogs, and by Bisgard and Musselman (1940) using goats, created a mild lateral curvature with minimal rotation. A similar outcome was achieved by Nachlas and Borden (1951) who then attempted to correct the deformity by a further operation injuring the other side of the growth plate. Of six dogs reported, two grew straight and two showed some improvement of the curvature. In all these efforts associated rotation of the vertebrae was either absent or minimal, and when Matzen (quoted by Wittebol, 1956) repeated the work of Nachlas and Borden he emphasized this.

Attention to the neurocentral junction and asymmetric pedicle growth as a cause of vertebral rotation was considered by Knutson (1963). He noted the observations of Nicoladoni (1909) who, on dissecting infantile scoliotic spines, found that the neurocentral junction was open on the concave part of the scoliotic curvature and closed on its convex part. Accordingly, when Ottander (1963) fused the neurocentral junction unilaterally in the second lumbar vertebra of the pig he produced a minimal scoliotic deformity with about 5° of rotation. Similar work has been carried out by Beguiristain *et al.* (1980), who have inserted screws across the neurocentral junction to produce premature growth arrest.

A number of other procedures have induced spinal deformities which are not relevant to the situation in idiopathic scoliosis. A thoracogenic type of scoliosis was produced in rabbits by Bisgard (1935) by creating a traumatic pleurisy. Holding growing rabbits in a laterally flexed position by plaster casts has similarly created lateral spinal curvatures (Hakkarainen, 1975). Metabolic methods include the use of rachitic diets or alternatively diets containing beta-aminopropionitrile (the active agent of lathyrism) (Yamamoto, 1966; Lalic and Angevine, 1970). Finally, by persistent inbreeding, a strain of Leghorn chickens has been developed in which 50% of sexually mature animals develop a scoliotic deformity (Riggins *et al.*, 1977). Analysis of the collagen in these animals has shown similarities to the collagen from birds made mildly lathyratic.

BIOMECHANICS OF IDIOPATHIC SCOLIOSIS

The literature on the biomechanics of scoliosis is limited, but nevertheless some interesting observations have been made. An important factor in the kinematics or

dynamics of the vertebral column is the observation of coupling. This is applied to motion in which rotation or translation about or along one axis is consistently associated with rotation or translation about or along a second axis. In a classic study on the kinematics of the normal spine as related to scoliosis, Lovett (1905) showed that lateral deviation of the spine was associated with rotation of the vertebral bodies about a vertical axis. He showed that lateral bending in flexion produced rotation of the vertebral bodies to the convex side, whereas side bending in extension caused concave side rotation. In a more thorough study White (1969) demonstrated coupling in the thoracic spine and noted that in the upper portion there was a relatively marked and consistent coupling of axial rotation with lateral bending. However, the direction of coupling is such that the vertebral bodies rotate towards the concavity. In the middle and lower regions of the thoracic spine he found that the same pattern was still present, though neither so prominent nor consistent. Of interest he noted that the direction of coupling in the mid-thoracic region was occasionally reversed such that the vertebral bodies were directed towards the convexity. White goes on to consider that if some factor is to influence this coupling such that convex side rotation occurs then the elements of a scoliotic deformity might follow.

Using computational techniques and a knowledge of the normal motion capabilities of elements of the vertebral column Schultz *et al.* (1972) have analysed the changes necessary to convert a straight vertebral column into a scoliotic configuration. Not only did they find that lateral asymmetry of the vertebrae was not required to produce a scoliotic curve, but more particularly that mild scoliotic configurations were achievable within the motion capabilities of the normal spine. In order to achieve a scoliotic curve they inferred that a relative tethering of the extreme posterior structures was an important mechanism. The concepts of both White and Schultz can be unified if one considers in anatomical terms the vertebral rotation that occurs in scoliosis. In these curves it is seen that the vertebral bodies are always rotated to the convexity and the posterior elements into the concavity. This is effectively a rotated lordosis since rotating a kyphotic region about its vertical axis would bring the posterior elements on to the convexity. This altered or convex side vertebral body rotation could occur if a lordotic region existed within the thoracic spine and would effectively have arisen by a tethering of the posterior structures in this region. The idea of such a relatively lordotic region in the thoracic spine representing an area of asymmetry in the median plane will be seen to be fundamental to the pathogenesis of idiopathic scoliosis.

Concept of median plane asymmetry

It is well recognized that when the back of the idiopathic scoliosis patient is inspected there is, in addition to the lateral curvature and rotation, flattening of the normal thoracic kyphosis, particularly in the region of the curve apex (*Figure 1.6*). Furthermore, a study of cadavers with severe deformities has shown that this lordotic segment persists, with the effect of posterior tethering being such that the spinous processes are pulled back to a straight line (Dickson *et al.*, 1983).

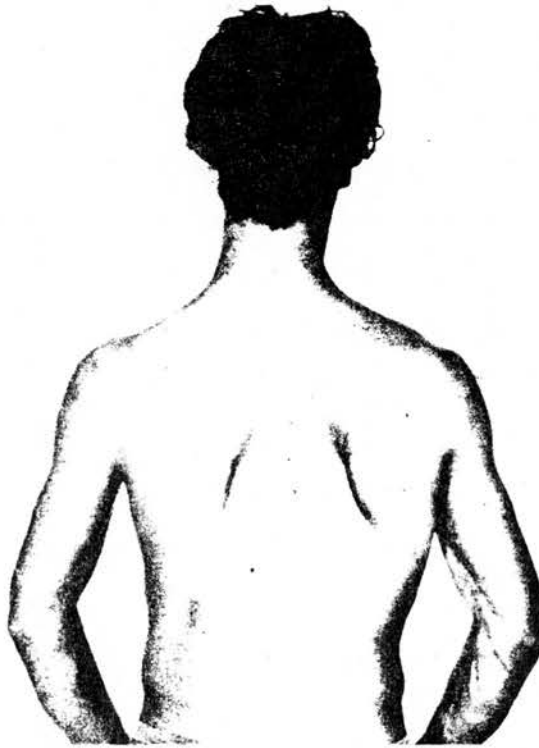


Figure 1.6 Back of a patient with a 40° right thoracic idiopathic scoliosis; there is a lordosis at the curve apex

Furthermore, whereas the lateral curvature of the spine receives the greatest attention when anteroposterior (AP) radiographs are taken, it is the fundamental vertebral rotation which allows an early scoliotic deformity to be detected. More significantly, the standard method of discerning such rotation is to bend the patient forward (Figure 1.7b), whereupon the rotational prominence becomes maximal, and indeed this is the basis of the forward bending test for the early detection of scoliosis. On resumption of the erect posture the rotation diminishes and, coupled with the tendency for the tips of the spinous processes to be brought back to the midline, means that an examination of the back in the erect posture may overlook the deformity.

Such behaviour of the curvature is explained by the concept that there is a region of median plane asymmetry at the curve apex such that the front of the spine is longer than the back. To accommodate this increased anterior height with forward flexion there must either be increased flexion above and below the segment or alternatively this region must twist out to the side. Such a tendency to rotational instability would be provided by any additional asymmetry in coronal or transverse planes. An important corollary of this is that if, during growth, the spine continues to grow faster at the front than at the back, then likewise rotation of this area of median plane asymmetry will be expected to occur.

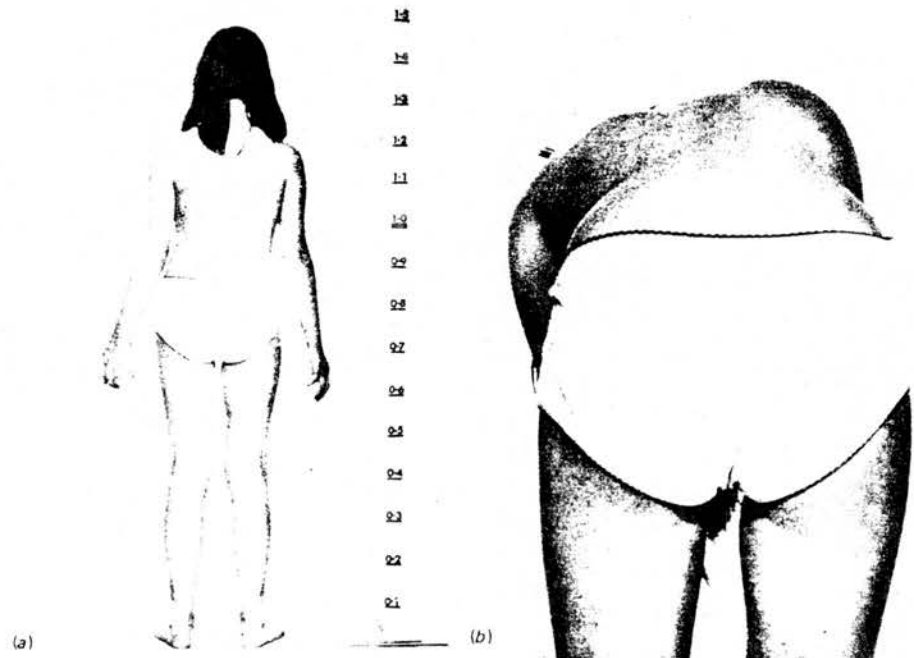


Figure 1.7 (a) The spine is unremarkable in the erect position. (b) Forward bending produces a significant rotational prominence

The concept of a primary lordosis is of course well established. First proposed by Adams in 1865, who stated that lordosis plus rotation equals lateral flexion, it received further attention from Heuer in 1927 and later by Somerville (1952) and Roaf (1966). Forward flexion of simple structural models with a stiff lordotic region allowed the simulation of a scoliotic deformity. Further work by Somerville using rabbit models was encouraging. His attempts at producing median plane asymmetry in the nature of a lordosis in the thoracic spine of rabbits with diathermy produced a progressive scoliosis with rotation in three animals. Unfortunately this promising work was not confirmed by Wittebol (1956) and Gottlieb (1957), who by producing a lordotic region only were disappointed to find that a progressive scoliosis did not ensue. This is because animals, unlike growing children, do not show any tendency to develop asymmetry in another plane required to provide the rotational instability. The inference from Somerville's work is that the insult he directed to the rabbit spine produced not only median plane asymmetry, but a few degrees of coronal plane asymmetry which allowed the system to spin and provided the necessary direction.

Concept of biplanar asymmetry

The concept therefore arises that the essential lesion of idiopathic scoliosis is median plane asymmetry (a lordosis or lordotic tendency) rendered unstable by asymmetry in the coronal or transverse planes. The former is required to produce a

situation prone to rotational instability and the latter to impart both initial momentum and direction. The mechanism is illustrated by *Figure 1.8*, a simplified diagram of the spine with pre-existing median and coronal plane asymmetry. Forward flexion of the relatively rigid lordotic segment produces a tightening of the posterior structures and their reactive force (f). The horizontal component of f acting at a distance (d) from the midline will impart a spinning moment (M) on the vertebral bodies with the spinous processes being drawn back to the midline (see

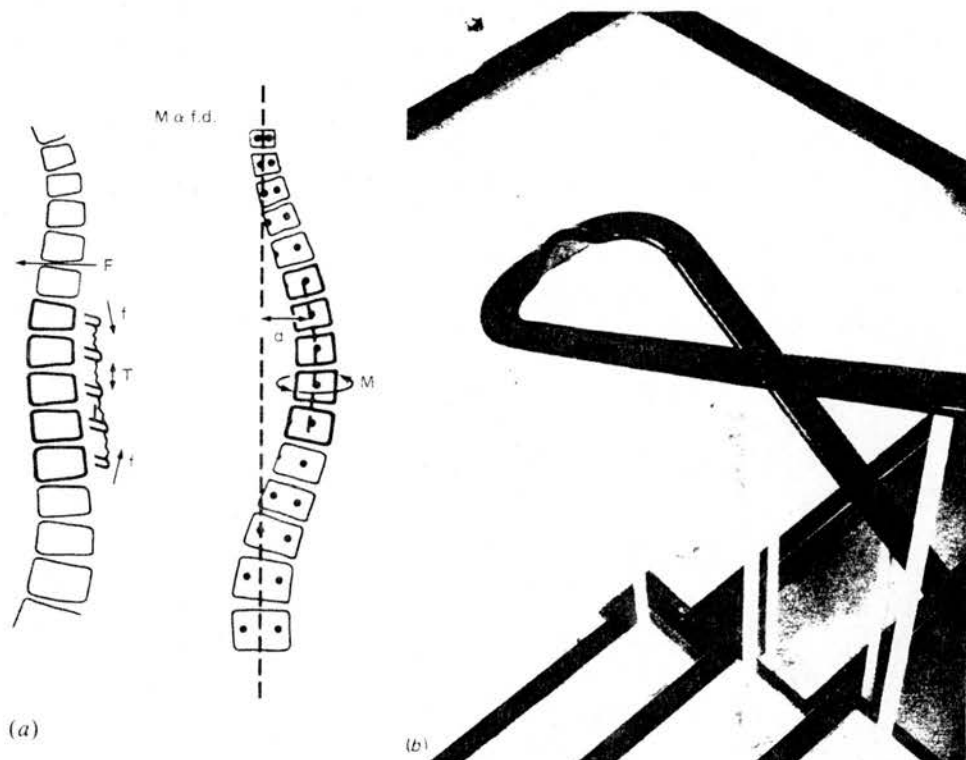


Figure 1.8 (a) Biomechanical model of a spine with biplanar asymmetry (lordosis plus coronal plane asymmetry) (courtesy of the Editor, *Journal of Bone and Joint Surgery*). For full explanation see text. *(b)* The mechanics of idiopathic scoliosis demonstrated by the bannister at St James's University Hospital, Leeds; at each landing the black plastic hand-rail covering is asked to describe a sharp U-turn. There is too much material on the concave side which must rotate to the side so that the curve can be accommodated

Figure 1.1). The magnitude of the spinning moment is therefore a function of the force of forward flexion and degree of coronal plane asymmetry. This rotational mechanism would be similar if the other asymmetry was in the transverse rather than coronal plane.

Our own dynamic studies of spinal motion in fresh cadaveric rabbit spines illustrate this mechanism. Forward flexion of the rabbit thoracic spine to the limits of normal motion is not coupled with any axial rotation (*Figure 1.9*). If, however, a

rigid lordotic segment is created in the thoracic spine and the specimen is flexed, then the relative tethering of the posterior structures creates a tendency for the spine to rotate, precipitated by any inherent coronal plane asymmetry in the system. The effect of rotation of this lordotic region is such that the vertebral bodies lie on the convexity of the curve. Excessive forward flexion of the normal spine beyond the limits of compression of the anterior structures does produce

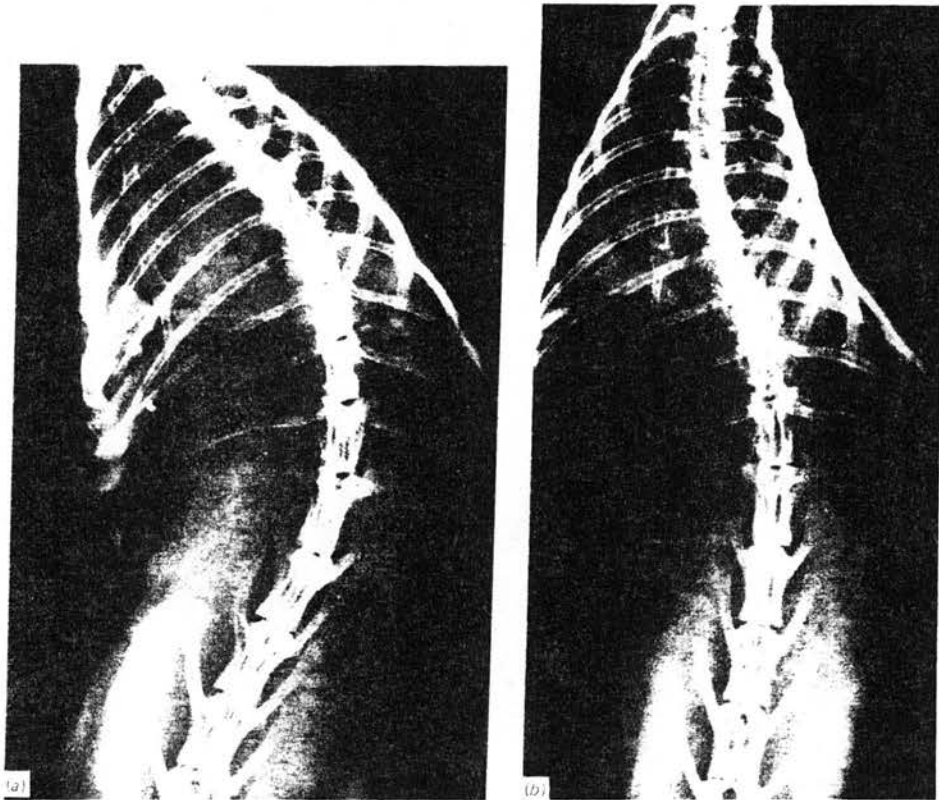


Figure 1.9 (a) When a normal fresh rabbit spine is flexed beyond its limits a lateral curvature develops but with the spinous processes directed towards the concavity as would be expected to occur with a rotated kyphosis. (b) When a similar preparation, but with a thoracic lordosis caused by posterior element approximation, is flexed a scoliosis occurs but with rotation such that the spinous processes are directed to the concavity as in idiopathic scoliosis (courtesy of the Editor, *Journal of Bone and Joint Surgery*) For a better quality picture see Publication 11, Figures 12 and 13.)

rotation, but with the bodies directed to the concavity as would be expected with a rotated kyphosis. In the absence of any abnormality of the posterior structures this relative tethering can be equally well achieved by comparative overgrowth of the vertebral bodies anteriorly, such that the spine is still lordotic and therefore unstable when full intersegmental flexion has occurred. It is interesting that the ability of such a spinal model to rotate does not depend upon the median plane asymmetry being solely in the nature of a true lordosis, as a flat segment or a

segment with a reduced kyphosis compared with above or below produces a similar effect.

How then do children develop this biplanar spinal asymmetry which produces their idiopathic scoliosis?

Coronal plane asymmetry in children

Worldwide epidemiological surveys derived from selective community screening have demonstrated enormous numbers of children, of the order of 15–25%, with coronal plane asymmetry of the spine although in only a few per thousand does curve magnitude achieve clinical significance (*see* Chapter 3). The great majority of these surveys have used the 1-minute school screening test as the first tier. This is a crude visual estimate of asymmetry of body topography and if the screening method were more sensitive then substantially more, and probably all, growing children would show evidence of coronal plane asymmetry (Avikainen and Vaherto, 1983). Indeed, if a scoliosis surgeon was presented with a spinal X-ray and a protractor he could probably find a scoliosis somewhere in the spine albeit of small magnitude. For that matter, there seems to be no inherent reason why a spine consisting of 17 thoracic and lumbar vertebrae piled on top of each other, separated by gristle and held up by guy ropes, growing in three dimensions simultaneously for at least 15 years in girls and 17 years in boys, should actually ever be straight, and in all probability it is not. Clearly another important factor or factors must be superimposed upon a minor coronal plane asymmetry for it to become a progressive idiopathic scoliosis.

Analysis of prevalence rates of coronal plane asymmetry by age and sex reveal a most interesting trend. For all ages coronal plane symmetry is less obvious in boys but, more importantly, the prevalence rate more than doubles between the ages of 12 and 13 in both boys and girls. It has already been shown that it is the overall environment of growth that appears to be of consequence rather than peak velocity in the production of a scoliosis, and the fact that something is happening to the adolescent spine in both sexes at the same time strongly suggests its independence of growth velocity, boys maturing on average about 2 years later than girls. Another major factor clearly influences the pre-existing coronal plane asymmetry at this time and acts in such a way that boys tend to be protected.

Transverse plane asymmetry in children

Vertebral anatomy in the transverse plane is critical in maintaining the integrity of spinal shape in the median plane. When a child is born there is a smooth kyphosis from foramen magnum to the lumbosacral junction which is lordotic at birth. When head control is developed by neck extension, a cervical lordosis appears and then when the child assumes the sitting and then standing positions the lumbar lordosis is fully developed. From an early age therefore there are four primary curves in the median plane, cervical and lumbar lordoses, and thoracic and sacral kyphoses. The shape of vertebrae in the transverse plane at different sites in the spine is important in understanding rotational instability.

In the thoracic region the vertebrae are heart shaped, shaped like a prism with the apex anteriorly (*Figure 1.10*). On flexion towards the apex this shape is rotationally unstable, exemplified by the fact that fishing rods triangular in section have been abandoned as they can only be cast towards the base. Accordingly this area of the spine is protected from rotation by an inbuilt thoracic kyphosis. The kyphosis is caused by the shape of the vertebrae in the median plane having reduced anterior compared with posterior height. Available intersegmental flexion is, however, small in the thoracic region and on flexion buckling will soon occur, producing an asymmetric kyphosis and a slight rib hump, but progressive rotation is prevented by the kyphotic shape. This is precisely what is being detected in so many

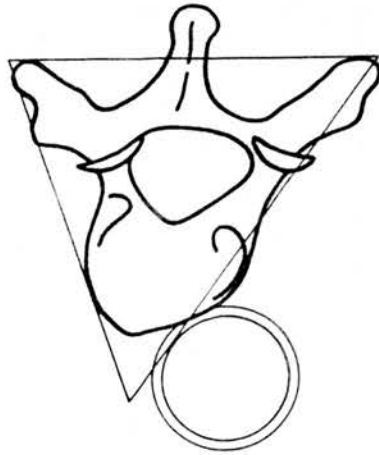


Figure 1.10 In the transverse plane the heart-shaped thoracic vertebrae are prismatic with the apex anteriorly but the prism is rendered asymmetric from D4 to D9 by the pressure effect of the left-sided aorta

normal children in the community (Burwell *et al.*, 1982). A minor rib hump appears on forward flexion but in the erect position the spine is perfectly straight in the coronal plane. Therefore these are not children with the mildest scoliosis that are being detected but a rotational kyphosis that is occurring on flexion in perfectly normal children. More importantly, the prismatic shape of the thoracic vertebral bodies is not symmetrical in the transverse plane. The bodies D4 to D8 are constantly flattened on the left side by the descending thoracic aorta (Inkster, 1953). On forward flexion this has the effect of rotating the apex of the prism to the right and explains why rib humps detected when normal children flex forwards have a 10:1 right:left ratio. The more pronounced the thoracic kyphosis, the more the aorta bowstrings across the vertebrae, losing contact with the left side of the prism and therefore less able or unable to produce the flattening effect. Therefore in children with a normal or pronounced thoracic kyphosis buckling can occur in either direction, whereas those whose thoracic kyphosis is flatter than normal will be more affected by the left-sided aorta and their kyphosis will buckle to the right. This is why rib humps in normal children have a 10:1 right:left ratio rather than the sort of ratio that would be anticipated by the rarity of situs inversus or dextrocardia. Rotational stability in the thoracic region depends therefore greatly on its inbuilt kyphosis and any significant reduction creates the necessary instability.

In contradistinction the cervical and lumbar regions would appear to be

particularly vulnerable to rotational instability, having inbuilt lordoses. However, the shape of the vertebral bodies in the transverse plane is an important protecting factor. In the cervical region the lateral to anteroposterior diameter has a ratio 3:2 and in the lumbar region this ratio is exceeded. The effect is therefore of a prism whose base is anteriorly directed, which confers great stability. In addition, intersegmental flexion in both cervical and lumbar regions is greatly in excess of that occurring in the thoracic region, such that the rotational instability of relative posterior tightness does not occur. Furthermore, both lordotic areas are supported by powerful muscular, fascial and ligamentous systems maintaining their integrity. However, it is well known in neuromuscular conditions in particular that any asymmetric failure of the soft tissue supporting system renders the lordosis immediately vulnerable to rotation.

Interestingly, while the thoracic region is arbitrarily defined as extending from D1 to D12, this is not so anatomically (Inkster, 1953). In both median and transverse planes the first two and last three thoracic vertebrae resemble typical cervical and lumbar vertebrae respectively. They are therefore downward extensions of the cervical lordosis and upward extensions of the lumbar lordosis respectively. Thoracic idiopathic curves are bimodal in their frequency pattern as regards site. By far the commonest site is a curve apical at D8/9 while a second and much less common site is apical at D3. When a lordosis or lordotic tendency occurs in these two regions then the prismatic-shaped thoracic vertebrae with the apex anteriorly are no longer protected by a kyphosis and immediately rotate to the side governed by the closer position of the descending aorta. The aorta loses its left-sided position as it descends, becoming midline over D12. The preponderance of right-sided curves in the thoracic region diminishes the lower the curve apex until left-sided lumbar curves are more common. In the lumbar region the abdominal aorta moves to the left of the midline as it approaches its bifurcation and now provides a dynamic form of transverse plane asymmetry such that there is a preponderance of left-sided curves in the lumbar region (*Figure 1.11*). These transverse plane asymmetries are crucial for the development of both rib humps in normal children and minor degrees of coronal plane asymmetry with rotation, which are both observed in such large numbers when children are screened.

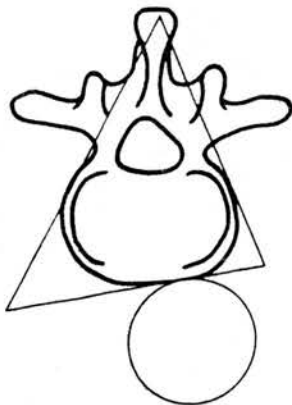
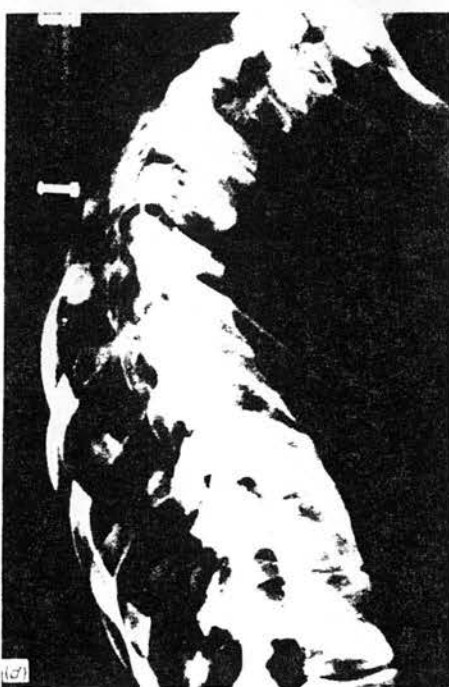


Figure 1.11 In the transverse plane the lumbar vertebrae are also prismatic but with the base anteriorly; the left-sided abdominal aorta facilitates left-sided rotation



(For a better quality picture see Publication 11, Figures 5 to 10.)

Median plane asymmetry in children

Since Adams in 1865 this has failed to receive the attention it deserves because of the general obsession with the lateral curvature of the spine as observed on an anteroposterior radiograph of the patient. Furthermore, this lordotic region soon becomes disguised clinically by the development of a rib hump, secondary to vertebral rotation, and the apparent development of a 'kyphoscoliosis'. Because of the vertebral rotation, inherent in any idiopathic scoliotic deformity, a lateral radiograph of the patient will not encompass a lateral view of the apex of the curve and therefore will mask any median plane asymmetry. To obtain a true lateral view of the apex it is necessary to rotate the patient to the same extent as the apical vertebral rotation. Clinically this can be achieved by taking a view tangential to the rib hump, though accuracy is improved by preliminary fluoroscopic screening or scanning of the spine. When this is performed on children with small idiopathic curvatures, over 80% of the cases show a lordotic region at the apex, and in the remainder the median plane curvature of this region is flatter than the kyphotic area immediately above (Dickson *et al.*, 1983).

The situation is still maintained when spines with more severe curvatures are examined (Deane and Duthie, 1973). Furthermore, an analysis of such a spine illustrates the fallacy of the so-called 'kyphoscoliotic' spine and illustrates the need to see the spine in three dimensions. *Figure 1.12* is a photographic and radiographic composite which explains the illusion. Because of the associated vertebral rotation, severe curves, when viewed in relation to the patient, are directed not only



Figure 1.12 The kyphoscoliosis illusion: (a) A specimen of idiopathic scoliosis viewed from the front. (b) Anteroposterior radiograph of specimen showing the scoliosis. (c) Specimen viewed from the side showing an apparent kyphosis. (d) Lateral radiograph showing the same illusion. (e) True lateral view of curve apex showing a flat segment. (f) True lateral radiograph of curve apex showing a lordosis

sideways but also backwards. Thus when the patient is viewed from the front a lateral curvature of the spine is seen but this does not represent the true severity of the curve as it is not seen *en face*. Indeed, the X-ray looks more like a lateral of the curve apex than an anteroposterior projection, which in fact it nearly is. Because of the posterior directed curvature, the lateral view of the patient gives the appearance of a kyphosis. However, the accompanying radiography shows that this is in

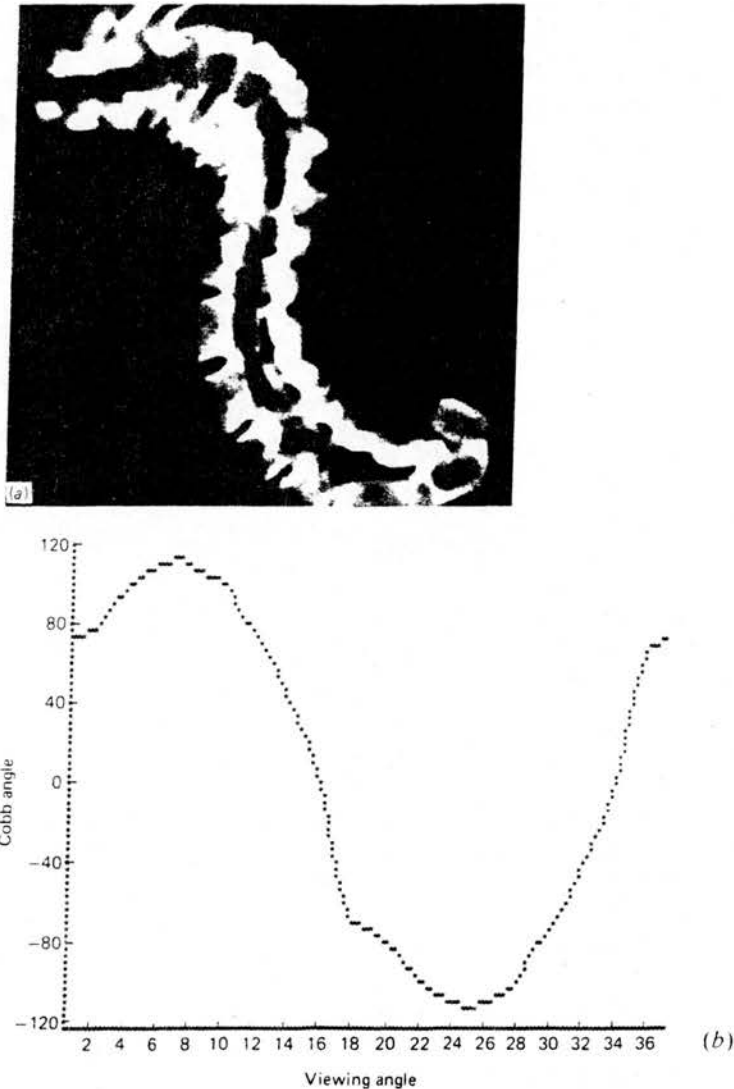


Figure 1.13 (a) Longitudinal CT scan of same specimen as in Figure 1.12 (12 mm thick) showing an entire spine on one slice; a kyphosis cannot exist (courtesy of the Editor, *Journal of Bone and Joint Surgery*). (b) Graph showing how Cobb angle changes with the direction of the X-ray beam

fact the scoliosis itself now seen in an almost anteroposterior view. It is apparent that the same single curvature is being viewed in two planes at right angles, neither view being a true anteroposterior or lateral of the curve. Despite the clinical appearance of a kyphosis, the median plane asymmetry persists with a small lordosis at the curve apex measuring only 5° when a true lateral projection of the apex is obtained. The absence of any significant curvature of the spine in two orthogonal planes is evidenced by the fact that the entire spine can be visualized on one longitudinal CT scan only 12 mm thick (*Figure 1.13*). Therefore idiopathic kyphoscoliosis cannot and does not exist.

Further evidence regarding median plane asymmetry is contained in the work of Willner (1981), who assessed the lateral profile of the spine of children. Comparing normal children with those with idiopathic scoliosis and those with Scheuermann's disease he determined that those with idiopathic scoliosis had the flattest overall thoracic kyphoses (*Figure 1.14*). Equally significant though were his observations

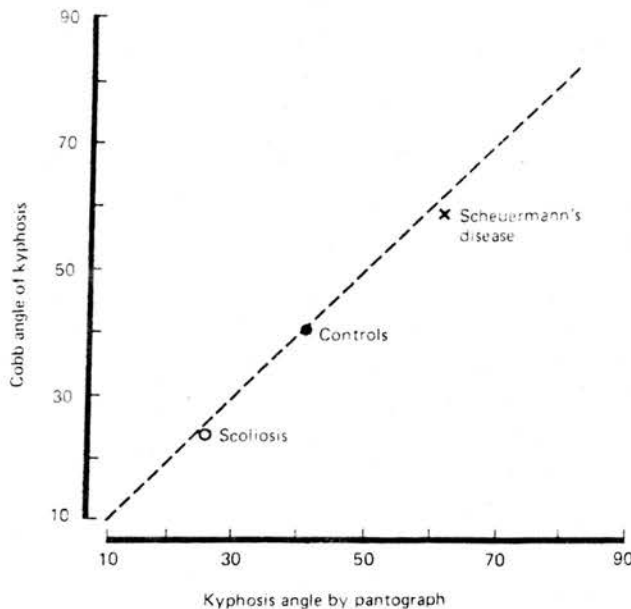


Figure 1.14 Magnitude of thoracic kyphosis in idiopathic scoliosis, normal children and Scheuermann's disease (after Willner, 1981)

on the change in thoracic kyphosis with growth. As children enter adolescence so their thoracic kyphosis reduces to a minimum, occurring just beyond the age of 10 years simultaneously in boys and girls. However, this thoracic kyphosis flattening is more obvious and maintained longer in girls than in boys. Unfortunately these observations involved assessing the overall kyphosis from top to bottom of the thoracic spine and did not reveal exactly where this flattening of the kyphosis is occurring. What is really being observed are the effects of the development of a relatively lordotic segment in the lower thoracic spine.

If screening then suggests that the development of coronal, transverse, and median plane spinal asymmetry is part of the normal development of the adolescent spine, then why should a progressive scoliotic deformity develop?

The effect of growth

The effect of the adolescent growth spurt on the progression potential of a minor scoliotic deformity is well recognized, but that girls with idiopathic scoliosis tend to be taller than their straight-backed counterparts is undoubtedly due to flattening of the primary curves in the median plane. The overall environment of growth is the

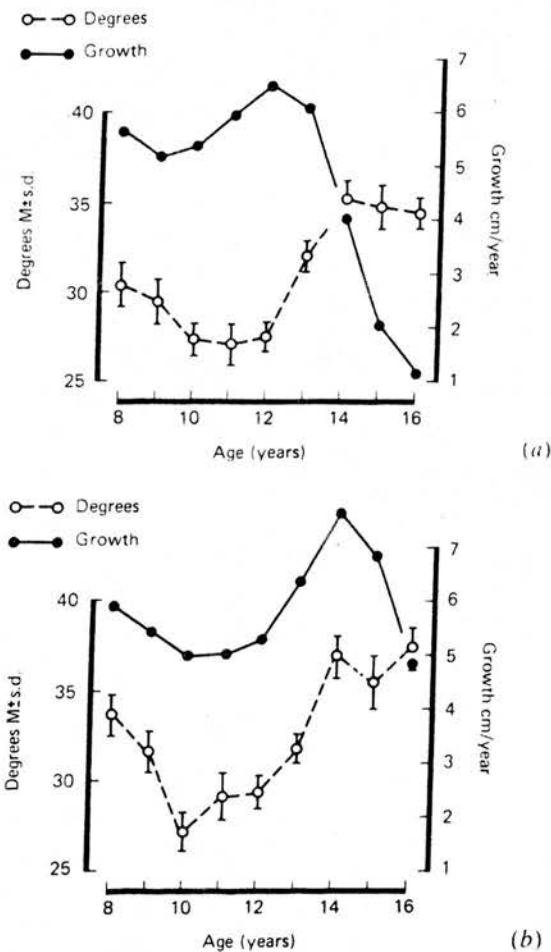


Figure 1.15 The thoracic kyphosis reduces in both girls and boys to a minimum between ages 10 and 12 years, but in girls (a) growth velocity is increasing, while in boys (b) growth velocity is constant (after Willner, 1981)

final factor in the pathogenesis of idiopathic scoliosis and the timing of this growth spurt in relationship to the development of median plane asymmetry explains the established clinical knowledge regarding progression potential in girls compared with boys. If the change in the thoracic kyphosis with age as noted by Willner is plotted against the growth velocity for girls and boys then the situation becomes clear (*Figure 1.15*). As girls achieve the maximum flattening of their thoracic kyphosis and are most susceptible to the inherent instability of the spine, they are entering their maximum growth spurt. This sets the scene for the situation to progress. However, with boys the growth spurt occurs on average 2 years later, at a time when their thoracic kyphosis is increasing again. Hence the growth spurt is occurring at a time when the spine is recovering its stability to rotation. However, the increased anterior growth plate loading that results, coupled with the influence of increased growth, would explain the susceptibility of such spines to develop an increased kyphosis or Scheuermann's disease, the opposite lesion to idiopathic scoliosis.

In summary, we have three important influences involved in the pathogenesis of idiopathic scoliosis as represented by the Venn diagram (*Figure 1.16*). The development of median plane asymmetry in association with inherent pre-existing

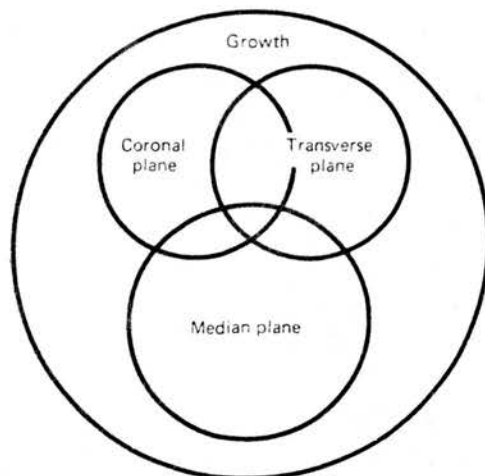


Figure 1.16 Venn diagram illustrating the important factors in the pathogenesis of idiopathic scoliosis

coronal or transverse plane asymmetry creates a spinal column with marked rotational instability particularly on forward flexion. If this occurs in the overall environment of growth then a progressive deformity may develop with secondary growth deformity following asymmetric growth plate loading. The latter accounts for some lateral wedging of the vertebral bodies and asymmetric pedicle lengths apparent in the established scoliotic deformity. It is the attention to the secondary deformities which has for so long prevented appreciation of the essential lesion of progression idiopathic scoliosis.

Experimental progressive scoliosis

The majority of previous attempts at creating a scoliotic deformity in the experimental animal have concentrated on attempts to reproduce the secondary deformities of the established curve. Division of muscle, nerve, ligament or bones has produced paralytic or traumatic types of scoliosis with a lateral curvature but little or no rotation. Similarly, unilateral damage to the vertebral body growth plate or neurocentral junction has failed to produce a deformity akin to the clinical situation. Those workers who have appreciated the importance of median plane asymmetry have also been unrewarded in their experimental attempts. This is because animals, unlike man, have no pre-existing transverse or coronal plane asymmetry required in simple terms to start the system spinning. *Figure 1.17* is an anteroposterior and lateral radiograph of a rabbit spine in which a short-segment lordosis has been created in the lower thoracic region by approximating the spinous processes with wire. Despite 6 months of growth the spine remains disappointingly

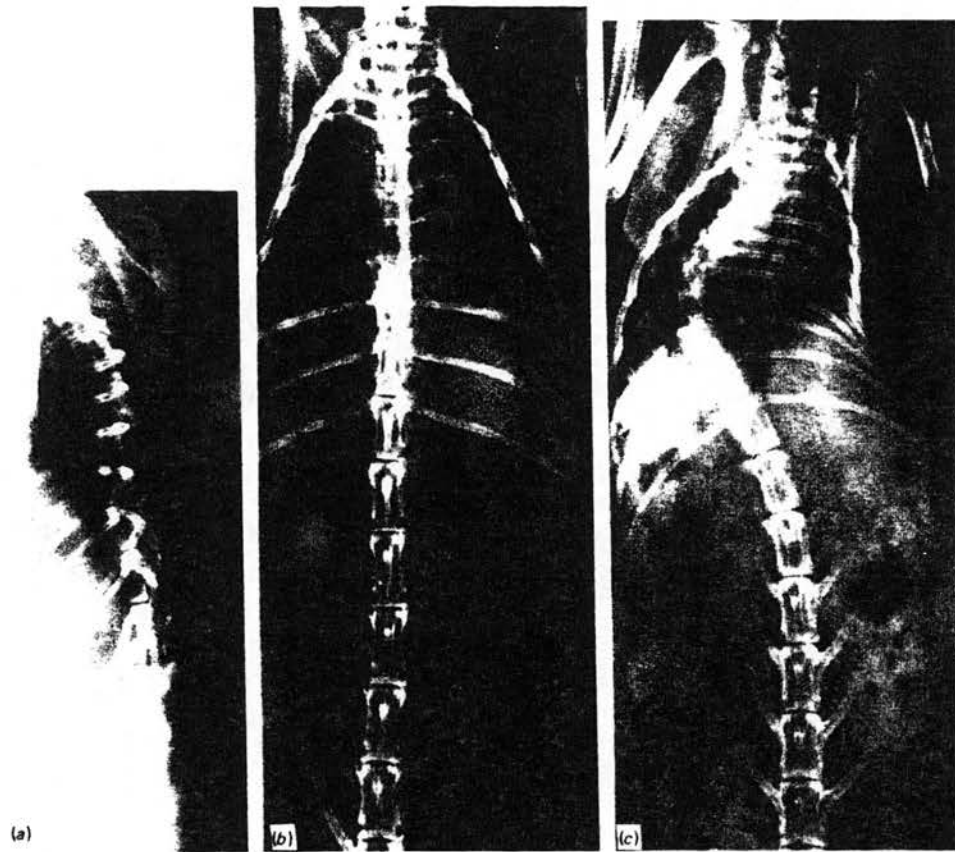


Figure 1.17 Experimental idiopathic scoliosis in the rabbit: (a) if a thoracic lordosis only is created then (b) the spine remains straight with growth; (c) if median and coronal plane asymmetry are both created then with growth an idiopathic-type deformity is produced (For a better quality picture see Publication 22, Figures 1 and 2.)

straight. When, however, synchronous biplanar asymmetry has been created then rotation of the vertebral bodies and a lateral curvature subsequently develop with growth, the scoliotic deformity progressing from week to week.

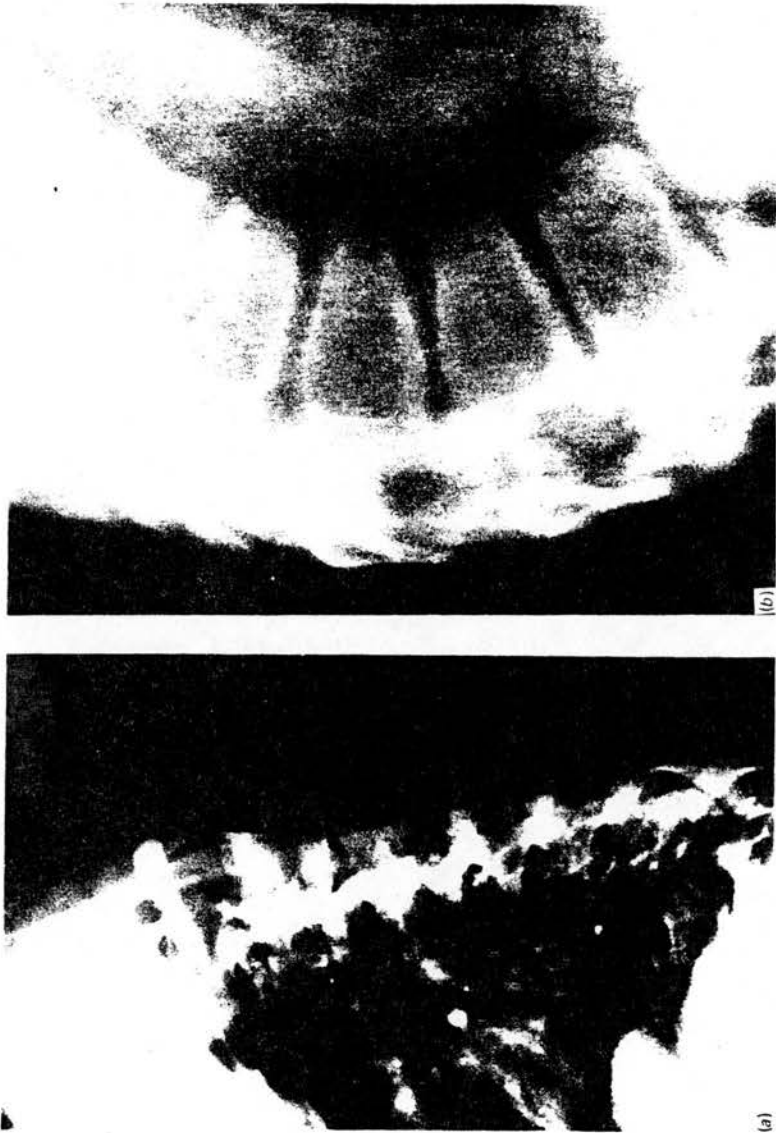
The underlying pathology

The clinical and biomechanical evidence for the underlying importance of median plane asymmetry is readily apparent. Furthermore, it can be seen that we are dealing with a situation which is part of the spectrum of normal development of the spine; but, when an unfortunate combination of biplanar asymmetry and rapid growth occurs, a progressive scoliotic deformity may develop. An underlying 'pathology' is therefore an incorrect term as this represents the extremes of the normal spectrum of development of the spine. This becomes most apparent when the radiological abnormalities in the median plane of the early scoliotic deformity are compared with Scheuermann's disease. The lateral radiograph of the apex of the scoliotic curve, *Figure 1.18*, shows that not only are the vertebral bodies wedge-shaped in the opposite direction to Scheuermann's disease, but that there is evidence of endplate irregularity towards the posterior aspect of the vertebral growth horseshoe (Deacon, Archer and Dickson, 1983; Roth, 1982). Conversely, with Scheuermann's disease the wedging and growth-plate irregularity occur anteriorly. This lends strong support to the concept of idiopathic scoliosis being a vertebral growth disorder or 'osteochondrosis of the spine', where growth-plate irregularity is tending to affect the posterior rather than anterior aspect of the vertebral body. Hence a specific aetiology does not have to be proposed. What is being observed are the ends of a spectrum of vertebral growth variation.

The concept of a spectrum of lateral profiles from flat through normal to those associated with Scheuermann's disease, as indicated in *Figure 1.14*, receives considerable support from epidemiological and genetic considerations. Scheuermann's disease has a similar community prevalence to idiopathic scoliosis and also has a familial trend (Sorenson, 1964). Furthermore, it is modal as regards site at the junction of the upper two-thirds and lower third of the thoracic spine, as is idiopathic scoliosis. The familial trend of idiopathic scoliosis, but as yet not elucidated mode of inheritance, probably reflects no more than a familial trend for a certain lateral profile, round backs come from round-back families and flat backs come from flat-backed families. It is therefore not the condition which is genetic or familial but the trend shared by families to have a spine vulnerable to rotation.

Biplanar asymmetry and the clinical behaviour of idiopathic scoliosis

Biplanar asymmetry of the spine explains the clinical features of idiopathic scoliosis. Structural curves exhibit increased rotation on forward flexion, while non-structural curves do not as they do not have median plane asymmetry. Braces or casts function primarily by preventing forward spinal flexion. Underarm orthoses are only effective with a curve apex not higher than D8 because, if the curve is higher, flexion cannot be prevented. Once the curve gets much beyond 30° it is vulnerable to gravity as a further deforming force and therefore merely the prevention of flexion may not prevent curve progression. An important aspect of a



(a) *Figure 1.18* Lateral spinal radiographs: (a) Idiopathic scoliosis – there is a lordosis and the apical vertebrae are wedged, being longer anteriorly. (b) Scheuermann's disease – there is a kyphosis and the apical vertebrae are wedged, being longer posteriorly (For a better quality picture see Publication 11, Figures 2 and 3.)

satisfactory brace, and that pointed out by Blount (Blount and Moe, 1973) when he first designed the Milwaukee brace, is that the lumbar lordosis should be obliterated. He found that both the Cobb angle and the amount of rotation were much improved when the lordosis was obliterated. This is also an important feature of casts or underarm braces. When the lordosis is obliterated, in order to stand erect the thoracic spine must compensatorily hyperextend. This provides more room up front for the vertebral bodies at the apex of the curve and therefore produces a derotation effect (*Figure 1.19*). In lumbar curves rotation is more obvious because there is already a lordosis in this area.

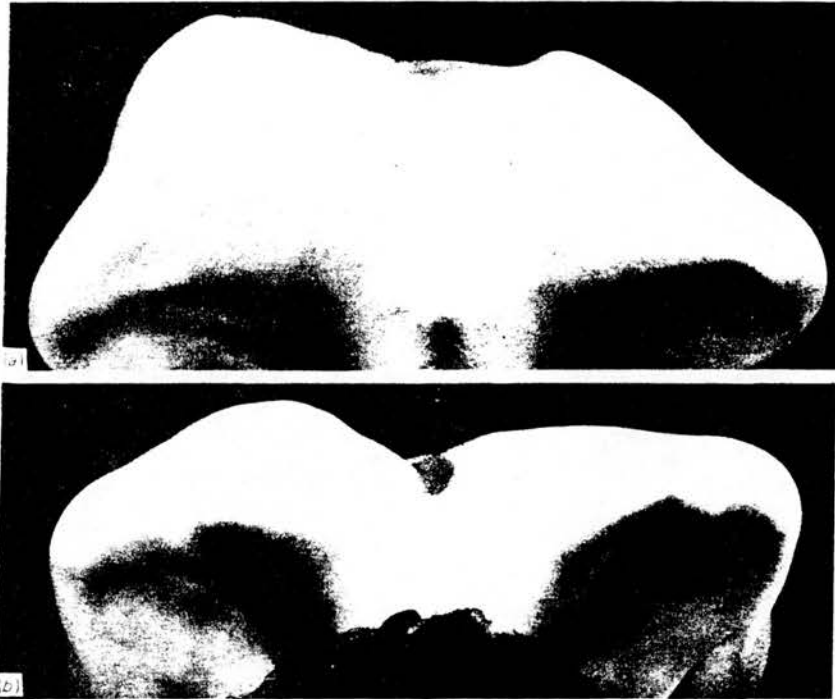


Figure 1.19 The effect of obliteration of the lumbar lordosis. (a) View from above of a right thoracic scoliosis and rib hump. (b) When the lumbar lordosis is obliterated thoracic hyperextension causes derotation

While a satisfactory animal model has taken time to develop there has been a superb human model ever since scoliosis surgery was practised. Too short a fusion posteriorly not only fails to halt curve progression but may in fact lead to accelerated deterioration because of its tethering effect posteriorly (Roaf, 1966). In this situation the median plane asymmetry already present has been compounded by the surgeon and this is always prone to happen unless the fusion extends at least to neutral vertebra above and to neutral vertebra below. Indeed, so devastating can be the effect of too short a fusion posteriorly that a curve, albeit bad enough already, can be converted into a rapidly progressive malignant condition with such a severe rotational deformity that early death is unavoidable. *Figure 1.20* illustrates

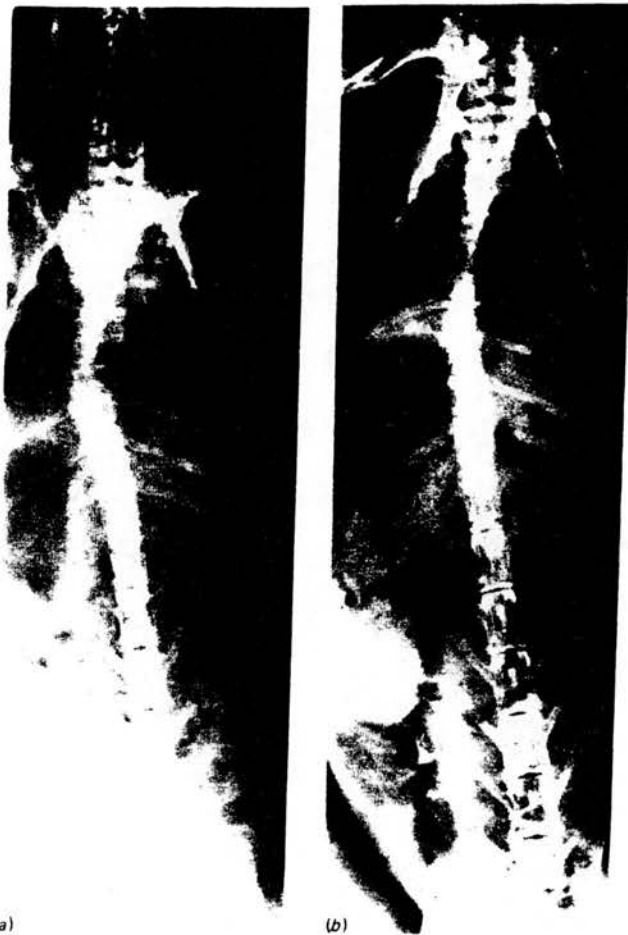


Figure 1.20 Experimental idiopathic scoliosis in the human. (a) A 56° left thoracic congenital scoliosis. (b) Three years after posterior fusion – the posterior tethering strut has produced severe progressive rotation and an idiopathic-type deformity (For a better quality picture see Publication 34, Figures 4 and 5.)

this point. At the age of 1 year plus 2 months this child had a 55° left thoracic congenital scoliosis with little, if any, rotation; a short posterior fusion *in situ* was performed at the age of 2 years. By the time she was 5 years 4 months the curve had progressed to 110° with severe rotation, an ugly chest deformity and significant reduction in pulmonary function. This required multiple posterior osteotomies, anterior vertebral body resection and posterior fusion with instrumentation (Leatherman and Dickson, 1979) to undo the damage that had been created by the previous operation.

The biological treatment of idiopathic scoliosis

Orthopaedic treatment of idiopathic scoliosis to date has concentrated on either attempting to hold an early deformity until growth of the spine is complete or alternatively, with more severe curves, obtaining a partial correction of the lateral



(a) (b)
 Figure 1.21 (a) Early idiopathic scoliosis in the rabbit. (b) When the thoracic kyphosis is recreated the spine grows straighter (For a better quality picture see Publication 22, Figure 3.)

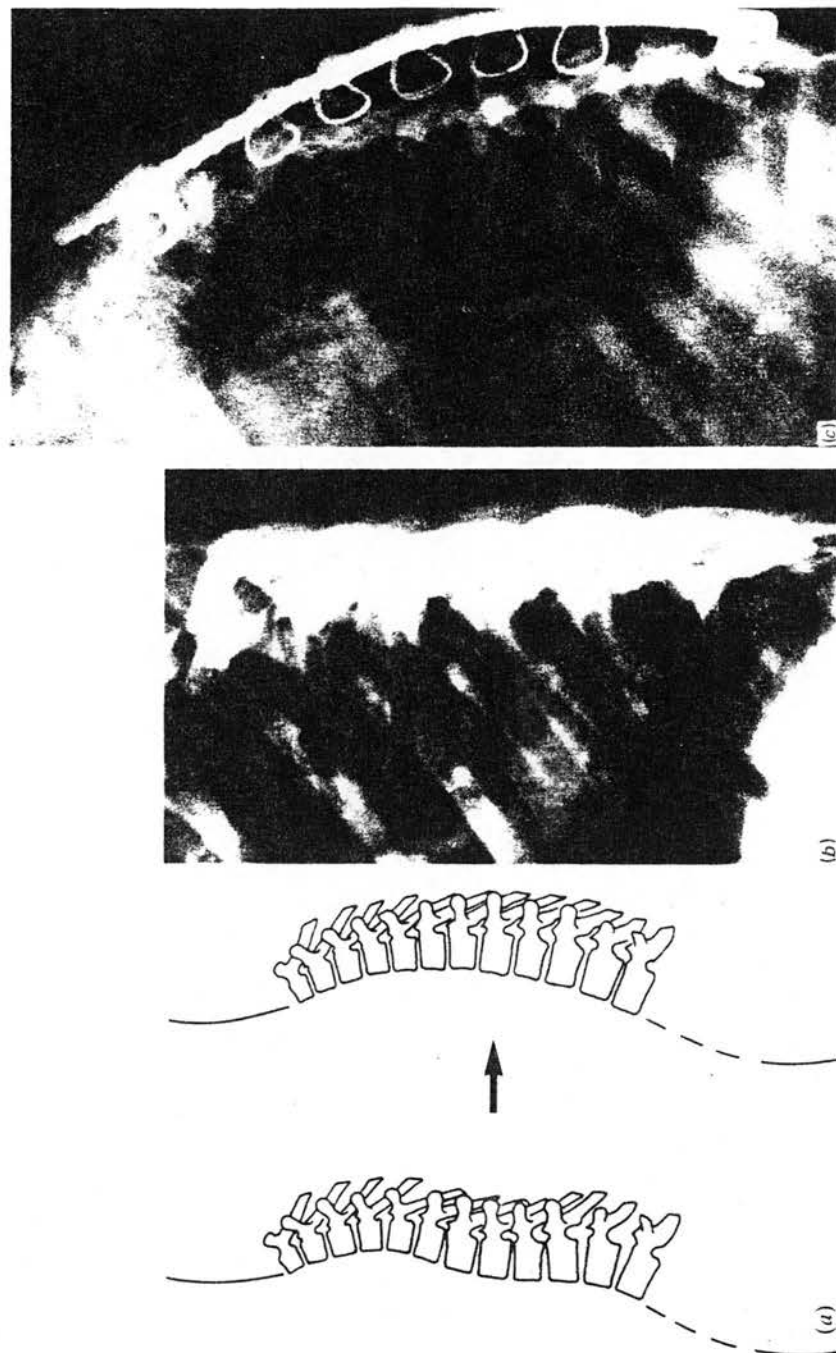
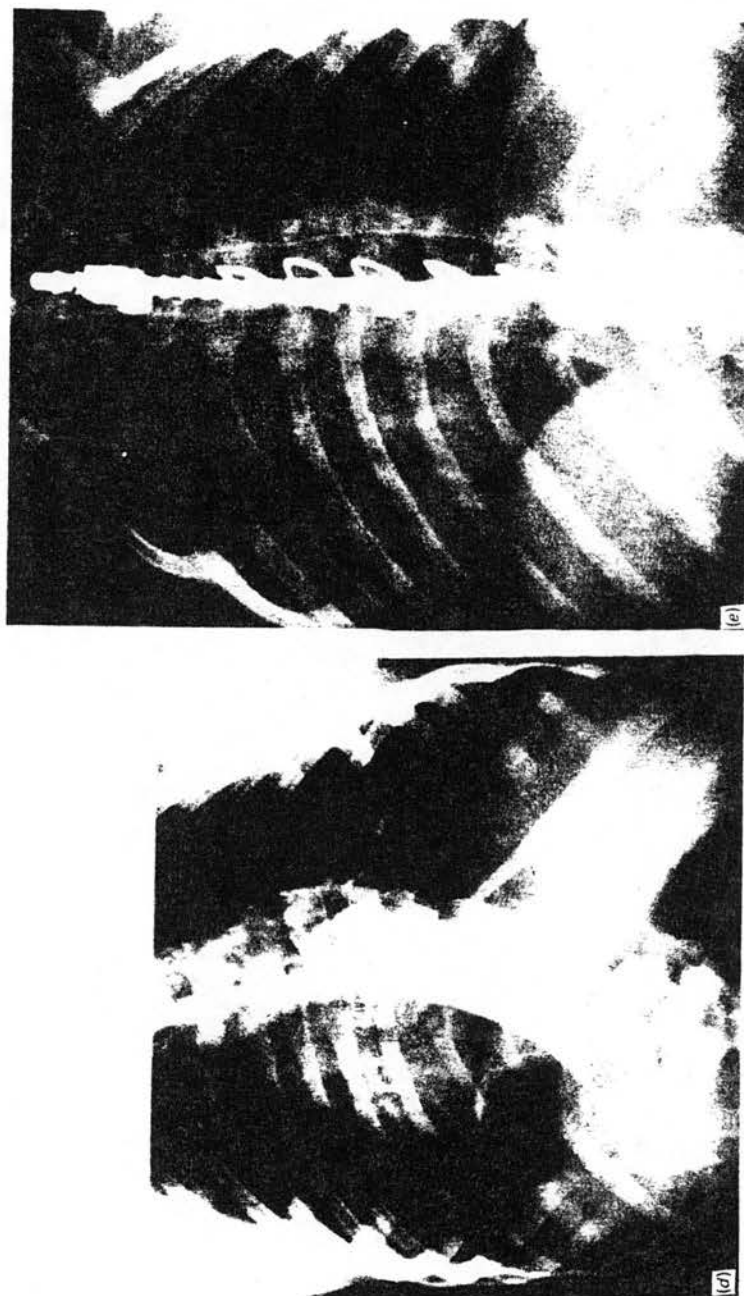


Figure 1.22 The biological treatment of idiopathic scoliosis. (a) Recreation of the normal thoracic kyphosis. (b) Lateral radiograph of a thoracic curve before treatment. (c) Lateral radiograph after recreation of the thoracic kyphosis. (d) Anteroposterior radiograph before treatment. (e) Anteroposterior radiograph after treatment - initial correction is better than conventional treatment and the spine has not been fused, allowing growth to further correct the secondary deformity

(For a better quality picture see Publication 28, Figure 6.)



(For a better quality picture see Publication 28, Figure 6.)

curvature with instrumentation and fusing the residual deformity in the hope that the situation will not relapse. However, the elucidation of the pathogenesis of idiopathic scoliosis allows treatment to be directed towards the underlying problem rather than the established secondary deformities.

The fundamental concept is that if the median plane asymmetry can be corrected (the normal thoracic kyphosis recreated), then the spine may be able to grow straight again, as most certainly occurs in the experimental animal (*Figure 1.21*). In this respect the powerful effects of growth can be harnessed to act in favour of the patient rather than, as in present practice, tending to negate any attempts at correction. Clearly the nearer maturity, the less growth can be harnessed in favour of the patient. More importantly, with bigger curves it may well be that the secondary deformity has become autonomous and relatively fixed, in which case there would be no substitute for an attempt to achieve the best possible correction of the secondary deformity. Nonetheless, few surgeons would always be happy about the amount of improvement of the rotational deformity of a curve measuring 60° or more which is merely treated conventionally by any form of instrumentation. Therefore, for such curves we have recently been aiming to reduce the secondary deformity and then correct the primary lesion such that any further growth may act in favour of the patient. The secondary deformity is corrected by the use of the traditional Harrington distraction rod. This, however, is bent to conform with the normal thoracic kyphosis. Then, by means of segmental wiring, drawing the laminae on the concave side up to the rod, the spine is derotated and the normal thoracic kyphosis re-established. This transfers the loading on the vertebral endplates from the back towards the front and thus with further growth the situation can only improve (*Figure 1.22*). Obviously to increase the chance of this treatment working it is necessary to detect the curvature at an earlier stage. Furthermore, the importance of examining the spine from the side cannot be overstressed, since it is the underlying median plane asymmetry which provides the progression potential and which requires correction if the spine is ever to grow straight again.

References

- Adams, W. (1865) *Lectures on the Pathology and Treatment of Lateral and Other Forms of Curvature of the Spine*. London: Churchill and Sons
- Alexander, M. A. and Season, E. H. (1978) Idiopathic scoliosis: an electromyographic study. *Archives of Physical Medicine and Rehabilitation*, **59**, 314–315
- Avikainen, V. J. and Vaherto, H. (1983) A high incidence of spinal curvature. A study of 100 young female students. *Acta Orthopaedica Scandinavica*, **54**, 267–273
- Beguiristain, J. L., De Salis, J., Oriafio, A. and Canadell, J. (1980) Experimental scoliosis by epiphysiodesis in pigs. *International Orthopaedics*, **3**, 317–321
- Belstead, J. S. and Edgar, M. A. (1978) Early detection of scoliosis. *British Medical Journal*, **2**, 937–938
- Bisgard, J. D. (1935) Experimental thoracogenic scoliosis. *Journal of Thoracic Surgery*, **4**, 435–442

- Bisgard, J. D. and Musselman, M. M. (1940) Scoliosis – the experimental production and growth correction; growth and fusion of vertebral bodies. *Surgery, Gynaecology and Obstetrics*, **70**, 1029–1036
- Blount, W. P. and Moe, J. H. (1973) *The Milwaukee Brace*. Baltimore: Williams and Wilkins
- Boas, F. (1932) Studies in growth. *Human Biology*, **4**, 307–350
- Bolk, L. (1923) The menarche in Dutch women and its precipitated appearance in the youngest generation. *Proceedings of the Academy of Science of the Amsterdam Secular Society*, **26**, 650
- Burwell, R. G., James, N. J., Johnson, F. and Webb, J. K. (1982) The rib hump score: a guide to referral and prognosis? *Journal of Bone and Joint Surgery*, **64B**, 248
- Butterworth, T. R. and James, C. (1969) Electromyographic studies in idiopathic scoliosis. *Southern Medical Journal*, **62**, 1008–1010
- Caulfield, J. B., Rebeiz, J. and Adams, R. D. (1968) Viral involvement of human muscle. *Journal of Pathology and Bacteriology*, **96**, 232
- Cowell, H. R., Hall, J. N. and MacEwen, G. D. (1972) Genetic aspects of idiopathic scoliosis. *Clinical Orthopaedics*, **86**, 121–131
- Czeizel, A., Bellyei, A., Barta, O., Magda, T. and Molnar, L. (1978) Genetics of adolescent idiopathic scoliosis. *Journal of Medical Genetics*, **15**, 424–427
- Deacon, P., Archer, I. A. and Dickson, R. A. (1983) The anatomy and biomechanics of spinal deformity. *Journal of Bone and Joint Surgery* (in press)
- Deane, G. and Duthie, R. B. (1973) A new projectional look at articulated scoliotic spines. *Acta Orthopaedica Scandinavica*, **44**, 351–365
- DeGeorge, F. V. and Fisher, R. L. (1967) Idiopathic scoliosis: genetic and environmental aspects. *Journal of Medical Genetics*, **4**, 251–257
- Dickson, R. A., Lawton, J. O., Archer, I. A. *et al.* (1983) Combined median and coronal plane asymmetry – the essential lesion of progressive idiopathic scoliosis. *Journal of Bone and Joint Surgery*, **65B**, 368
- Dickson, R. A. and Sevitt, E. A. (1982) Growth and idiopathic scoliosis: a longitudinal cohort study. *Journal of Bone and Joint Surgery*, **64B**, 385
- Duthie, R. B. (1959) The significance of growth in orthopaedic surgery. *Clinical Orthopaedics*, **14**, 7
- Duval-Beaupere, G. (1971) Growth and its relation to bone deformities. In *Scoliosis and Growth*, edited by P. A. Zorab, 58. Edinburgh and London: Churchill Livingstone
- Editorial (1975) Scoliosis. *British Medical Journal*, **2**, 661–662
- Faber, A. (1935) Skoliose bei eineiigen Zwillingen. *Der Erbarzt*, **2**, 102
- Faber, A. (1936) Untersuchungen ueber die Erbllichkeit der Skoliose. *Archiv für Orthopaedische und Unfallchirurgie*, **36**, 217–296
- Fidler, M. W., Jowett, R. L. and Troup, J. D. G. (1974) Histochemical study of the function of multifidus in scoliosis. In *Scoliosis and Muscle*, edited by P. A. Zorab, 184–192. London: William Heinemann Medical Books Ltd
- Fidler, M. W. and Jowett, R. L. (1976) Muscle imbalance in the aetiology of scoliosis. *Journal of Bone and Joint Surgery*, **58B**, 1–9
- Goodgold, J. (1974) *Anatomical Correlates of Clinical Electromyography*, 11. Baltimore: Williams and Wilkins

- Gorynski, T. and Bojkowa, M. (1957) Histological changes of spinal muscles in dystonic scoliosis. *Chirurgia Narzadow Ruchu Ortopedia Polska*, **22**, 139-143
- Gottlieb, O. (1957) Experimental scoliosis (advance report). *Acta Orthopaedica Scandinavica*, **26**, 295-296
- Haas, S. L. (1939) Experimental production of scoliosis. *Journal of Bone and Joint Surgery*, **21**, 963-968
- Hakkarainen, S. (1975) Growth disturbance of muscle - a possible factor in the pathogenesis of scoliosis. Experimental study. *Acta Orthopaedica Scandinavica*, **44**, 101-102
- Hassan, I. and Bjerkreim, I. (1983) Progression in idiopathic scoliosis after conservative treatment. *Acta Orthopaedica Scandinavica*, **54**, 88-90
- Henssge, J. (1964) Elektromyographischer Beitrag zum Skoliosenproblem. *Zeitschrift für Orthopaedie und ihre Grenzgebiete*, **99**, 167-195
- Henssge, J. (1968) Are signs of denervation of muscles of the spine primary or secondary findings in cases of scoliosis. *Journal of Bone and Joint Surgery*, **50B**, 882
- Heuer, F. (1927) Zur Theorie der Skoliose. *Hessisches Aerzteblatt*, no. 18 and ff. Quoted by Wittebol, P. (1956) Idiopathic scoliosis (experimental investigation). *Archivum Chirurgicum Neerlandicum*, **8**, 269-279
- Hewitt, D. (1957) Some familial correlations in height, weight and skeletal maturity. *Annals of Human Genetics*, **22**, 26-35
- Hirano, S. (1972) Electronmicroscopic studies on the back muscles in scoliosis. *Journal of the Japanese Orthopaedic Association*, **46**, 47-62
- Inkster, R. G. (1953) Osteology. In *Cunningham's Textbook of Anatomy*, 9th edition, edited by J. C. Brash. 136. London: Oxford Medical Publications
- James, J. I. P., Lloyd-Roberts, G. C. and Pilcher, M. F. (1959) Infantile structural scoliosis. *Journal of Bone and Joint Surgery*, **41B**, 719-735
- Kaneko, T. (1968) Histopathological and histochemical studies on the back muscles in scoliosis. *Journal of the Japanese Orthopaedic Association*, **42**, 13-28
- Khosla, S., Tredwell, S. J., Day, B., Shinn, S. L. and Ovalle, W. I. C. (1980) An ultrastructural study of multifidus muscle in progressive idiopathic scoliosis. *Journal of the Neurological Sciences*, **46**, 13-31
- Knutsson, F. (1963) A contribution to the discussion of the biological cause of idiopathic scoliosis. *Acta Orthopaedica Scandinavica*, **33**, 98
- Lalich, J. J. and Angevine, D. M. (1970) Dysostosis in adult rats after prolonged β -aminopropionitrile feeding. *Archives of Pathology*, **90**, 22
- Langenskiöld, A. and Michelsson, J. E. (1961) Experimental scoliosis in the rabbit. *Journal of Bone and Joint Surgery*, **43B**, 116-120
- Leatherman, K. D. and Dickson, R. A. (1979) Two-stage corrective surgery for congenital deformities of the spine. *Journal of Bone and Joint Surgery*, **61B**, 324-328
- Le Febvre, J., Triboulet-Chassevant, A. and Missirliu, M. F. (1961) Electromyographic data in idiopathic scoliosis. *Archives of Physical Medicine and Rehabilitation*, **42**, 710-711
- Lerique and Le Coeur (1951) Action potential asymmetry on two sides. Cited by Le Febvre, J., Triboulet-Chassevant, A. and Missirliu, M. F. (1961)

- Liszka, O. (1961) Spinal cord mechanisms leading to scoliosis. *Acta Medica Polona*, **2**, 45-63
- Lovett, R. W. (1905) Die Mechanik der normalen Wirbelsaeule und ihr Verhaeltnis zur Skoliose. *Zeitschrift für Orthopaedische Chirurgie*, **14**, 399
- Low, W. D., Mok, C. K., Leong, J. C. Y., Yau, A. C. M. C. and Lisowski, F. P. (1978) The development of Southern Chinese girls with adolescent idiopathic scoliosis. *Spine*, **3**, 152-156
- MacEwen, G. D. (1968) Experimental scoliosis. In *Proceedings of a Second Symposium on Scoliosis: Causation*, edited by P. A. Zorab. National Fund for Research into Crippling Diseases Monograph, 18-20. Edinburgh: Livingstone
- MacEwen, G. D., Bunnell, W. P. and Sriram, K. (1975) Acute neurological complications in the treatment of scoliosis. *Journal of Bone and Joint Surgery*, **57A**, 404-408
- Manning, C. W. (1968) Experimental scoliosis. In *Proceedings of a Second Symposium on Scoliosis: Causation*, edited by P. A. Zorab. National Fund for Research into Crippling Diseases Monograph, 11-14. Edinburgh: Livingstone
- Nachlas, W. and Borden, J. N. (1951) The cure of experimental scoliosis by directed growth control. *Journal of Bone and Joint Surgery*, **33A**, 24-34
- Nicoladoni, C. (1909) *Anatomie und Mechanismus der Skoliose*. Berlin and Vienna: Urban and Schwarzenburg
- Nordwall, A. and Willner, S. (1975) A study of skeletal age and height in girls with idiopathic scoliosis. *Clinical Orthopaedics*, **110**, 6-10
- Ottander, H. G. (1963) Experimental progressive scoliosis in a pig. *Acta Orthopaedica Scandinavica*, **33**, 91-99
- Peretti, G. and Velluti, C. (1969) La teoria neuro-muscolare nella etiopatogenesi della scoliosi giovanile. *Rassegna Medica Sarda*, **72**, 33-46
- Piggott, H. (1971) Posterior rib resection in scoliosis. A preliminary report. *Journal of Bone and Joint Surgery*, **53B**, 663-671
- Riddle, H. F. V. and Roaf, R. (1955) Muscle imbalance in the causation of scoliosis. *Lancet*, **1**, 1245-1247
- Riggins, R. S., Abbott, U. K., Ashmore, C. R., Rucker, R. B. and McCarrey, J. R. (1977) Scoliosis in chickens. *Journal of Bone and Joint Surgery*, **59A**, 1020-1026
- Riseborough, E. J. and Wynne-Davies, R. (1973) A genetic survey of idiopathic scoliosis in Boston, Massachusetts. *Journal of Bone and Joint Surgery*, **55A**, 974-982
- Roaf, R. (1966) The basic anatomy of scoliosis. *Journal of Bone and Joint Surgery*, **48B**, 786-792
- Robin, G. C. and Cohen, T. (1975) Familial scoliosis: a clinical report. *Journal of Bone and Joint Surgery*, **57B**, 146-147
- Robin, G. C. and Stein, H. (1975) Experimental scoliosis in primates - failure of a technique. *Journal of Bone and Joint Surgery*, **57B**, 142-145
- Roth, M. (1982) Idiopathic scoliosis and Scheuermann's disease: essentially identical manifestations of a neuro-vertebral growth disproportion. *Radiologia Diagnostica (Berlin)*, **22**, 380-391

- Sahlstrand, T. and Petruson, B. (1979a) A study of labyrinthine function in patients with adolescent idiopathic scoliosis. I. An electronystagmographic study. *Acta Orthopaedica Scandinavica*, **50**, 759-769
- Sahlstrand, T. and Petruson, B. (1979b) Postural effects on nystagmus during caloric labyrinthine stimulation in patients with adolescent idiopathic scoliosis. II. An electronystagmographic study. *Acta Orthopaedica Scandinavica*, **50**, 771-775
- Sahlstrand, T., Petruson, B. and Ortengren, R. (1979) Vestibulo-spinal reflex activity in patients with idiopathic scoliosis. *Acta Orthopaedica Scandinavica*, **50**, 275-281
- Schwartzmann, J. R. and Miles, M. (1945) Experimental production of scoliosis in rats and mice. *Journal of Bone and Joint Surgery*, **27**, 59-69
- Schultz, A. B., Larocca, H., Galante, J. O. and Andriecchi, T. P. (1972) A study of geometrical relationships in scoliotic spines. *Journal of Biomechanics*, **5**, 409-420
- Somerville, E. A. (1952) Rotational lordosis: the development of the single curve. *Journal of Bone and Joint Surgery*, **34B**, 421-427
- Sontag, L. W. and Nelson, V. L. (1933) A study of identical triplets. 1. Comparison of the physical and mental traits of a set of monozygotic dichorionic triplets. *Journal of Heredity*, **24**, 473-480
- Sorenson, K. H. (1964) *Scheuermann's Juvenile Kyphosis*. Copenhagen: Munksgaard
- Spencer, G. S. G. and Eccles, M. J. (1976) Spinal muscle in scoliosis. *Journal of the Neurological Sciences*, **30**, 143-154
- Spencer, G. S. G. and Zorab, P. A. (1976) Spinal muscle in scoliosis. *Journal of the Neurological Sciences*, **30**, 405-410
- Staub, H. A. (1922) Eine skoliotikerfamilie. Ein Beitrag zur Frage der kongenitalen Skoliose und der Hereditaet der Skoliosen. *Zeitschrift für Orthopaedie und Chirurgie*, **43**, 1-20
- Tanner, J. M. (1962) *Growth at Adolescence*, 2nd edition. Oxford: Blackwell Scientific Publications
- Tanner, J. M., Whitehouse, R. H. and Takaishi, M. (1966) Standards from birth to maturity for height, weight, height velocity and weight velocity. *Archives of Disease in Childhood*, **41**, 613-635
- Trontelj, J. V., Pecak, F. and Dimitrijevic, M. R. (1979) Segmental neurophysiological mechanisms in scoliosis. *Journal of Bone and Joint Surgery*, **61B**, 310-313
- Webb, J. N. and Gillespie, W. J. (1976) Virus-like particles in paraspinal muscle in scoliosis. *British Medical Journal*, **2**, 912-913
- Weiss, M., Milkowska, A. and Kozinska, M. (1957) Zachowawcze leczenie boczywnych skrzywien kregoslupa w swietle badan elektromiograficznych. *Chirurgia Narzadow Ruchu Ortopedia Polska*, **22**, 197-209
- White, A. A. (1969) Analysis of the mechanics of the thoracic spine in man. *Acta Orthopaedica Scandinavica*, Supplement 127, 1-105
- Willner, S. (1974) A study of growth in girls with adolescent idiopathic structural scoliosis. *Clinical Orthopaedics*, **101**, 129

- Willner, S. (1981) Spinal pantograph – a non-invasive technique for describing kyphosis and lordosis in the thoraco-lumbar spine. *Acta Orthopaedica Scandinavica*, **52**, 525
- Wittebol, P. (1956) Idiopathic scoliosis (experimental investigation). *Archivum Chirurgicum Neerlandicum*, **8**, 269–279
- Wong, Y. C., Yau, A. C. M. C., Low, W. D. and Lisowski, F. P. (1977) Ultrastructural changes of the back muscles of idiopathic scoliosis. *Spine*, **2**, 251–260
- Wynne-Davies, R. (1965) Familial scoliosis. In *Proceedings of a Symposium on Scoliosis* (London, 1965) National Fund for Research into Poliomyelitis and Other Crippling Diseases, 36. London: E. and S. Livingstone Ltd
- Yamada, K., Takaaki, I., Yamamoto, H., Nagawa, Y., Tanaka, H. and Tezuka, A. (1969) Equilibrium function in scoliosis and active corrective plaster jacket for the treatment. *Tokushima Journal of Experimental Medicine*, **16**, 1
- Yamada, K. and Yamamoto, H. (1972) Equilibrium function in scoliosis and active corrective plaster jacket for treatment. *American Digest of Foreign Orthopaedic Literature*, **3**, 182–185
- Yamamoto, H. (1966) Experimental scoliosis in rachitic bipedal rats. *Tokushima Journal of Experimental Medicine*, **13**, 1–34
- Yarom, R. and Robin, G. C. (1979) Studies on spinal and peripheral muscles from patients with scoliosis. *Spine*, **4**, 12–21
- Zuk, T. (1962) The role of spinal and abdominal muscles in the pathogenesis of scoliosis. *Journal of Bone and Joint Surgery*, **44B**, 102–105

IDIOPATHIC SCOLIOSIS IN THREE DIMENSIONS.

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As it is not ethically possible to obtain multiple radiographs, taken in small serial increments of rotation, of the deformed spines of otherwise normal children, articulated scoliotic spines from a pathological museum were studied. Eleven such specimens were xrayed incrementally through 180°. Vertebral body heights were measured anteriorly and posteriorly. This study demonstrated that the shape of the spine was entirely dependent on the radiographic projection and showed how uninformative are views taken antero-posterior and lateral with reference to the patient and not the deformity. The apical region of each curve was confirmed to be lordotic at bone level.

IDIOPATHIC SCOLIOSIS IN THREE DIMENSIONS

A RADIOGRAPHIC AND MORPHOMETRIC ANALYSIS

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Eleven articulated scoliotic spines were examined radiographically and morphometrically. Measurement of the curve on anteroposterior radiographs of the specimens gave a mean Cobb angle of 70°, though true anteroposterior radiographs of the deformity revealed a mean Cobb angle of 99° (41% greater). Lateral radiographs gave the erroneous impression that there was a mean kyphosis of 41° while true lateral projections revealed a mean apical lordosis of 14°.

Morphometric measurements confirmed the presence of a lordosis at bony level, the apical vertebral bodies being significantly taller anteriorly ($P < 0.02$). There were significant correlations ($P < 0.01$) between the true size of the lateral scoliosis, the amount of axial rotation and the size of the apical lordosis. This study illustrates the three-dimensional nature of the deformity in scoliosis and its property of changing in character and magnitude according to the plane of radiographic projection.

Although idiopathic scoliosis is defined as a lateral curvature of the spine with no associated neuromuscular condition or congenital spinal anomaly, the deformity is more complex and does not exist in one plane only. A lordosis rotates to one side, producing scoliosis as a secondary phenomenon; there is, therefore, deformity in all three planes (Adams 1865; Dickson *et al.* 1984).

Despite the triplanar nature of scoliosis, conventional attempts to quantify the deformity are based upon measurements made on radiographs taken in one plane only. The method described by Cobb (1948) is generally preferred to that of Ferguson (1930) and measures, on an anteroposterior radiograph of the patient, the angle subtended by lines drawn parallel to the upper and lower end vertebrae. In an effort to measure the amount of rotation at the apex of the curve, Cobb measured the distance that the apical spinous process was displaced towards the concavity; this was modified by Nash and Moe (1969) with reference to the pedicles.

Our knowledge of the natural history of the idiopathic scoliotic deformity and its response to treatment are based upon these uniplanar measurements. However, as the lordosis rotates to the side it becomes more posteriorly directed, so that measurements on an anteroposterior radiograph do not reveal the full extent of the

scoliosis. Only in one particular plane is the deformity truly seen, *en face*, and this has been referred to as the "plan d'élection" (du Peloux *et al.* 1965).

Three-dimensional analysis of spinal shape in scoliosis demonstrates the important relationship between the size of the scoliosis, the size of the lordosis and the amount of axial rotation, and indicates that the spinal deformity cannot be adequately quantified in one plane only.

MATERIAL AND METHODS

Eleven articulated spines with idiopathic scoliosis were studied; they were borrowed from the museum of the Royal College of Surgeons of Edinburgh.

Radiographic analysis. Each spinal column was mounted vertically on a specially constructed and calibrated turntable. Radiographs of each specimen were taken at 10° intervals of rotation through the complete range of 180°. From each radiograph the size of the deformity was measured using Whittle's adaptation of Cobb's method (Whittle and Evans 1979). Sequential changes in spinal shape were plotted graphically and the size of the deformity on true anteroposterior and lateral projections was correlated with axial rotation.

Morphometric analysis. The length of each entire articulated spinal column was measured both anteriorly, along a line joining the midpoints of the vertebral bodies, and posteriorly, along the line of the spinous processes, with a flexible rule and vernier calipers. In seven of the specimens measurements were also made of the anterior and posterior height of the individual vertebral bodies in the structural curve. The statistical significance of these measurements was determined using Student's *t*-test and by calculation of the correlation coefficient.

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RESULTS

Radiographic analysis. Figure 1 shows the change in Cobb angle of the deformity with rotation in Specimen 1; similar graphs were obtained for all the spines. The initial point on the graph represents an anteroposterior projection of the specimen; the apparent Cobb angle of the curvature increases with rotation until a maximum is reached in the position which represents a true anteroposterior view of the deformity. Thereafter, with continued rotation, the apparent size of the deformity diminishes until it becomes minimal at a point 90° further

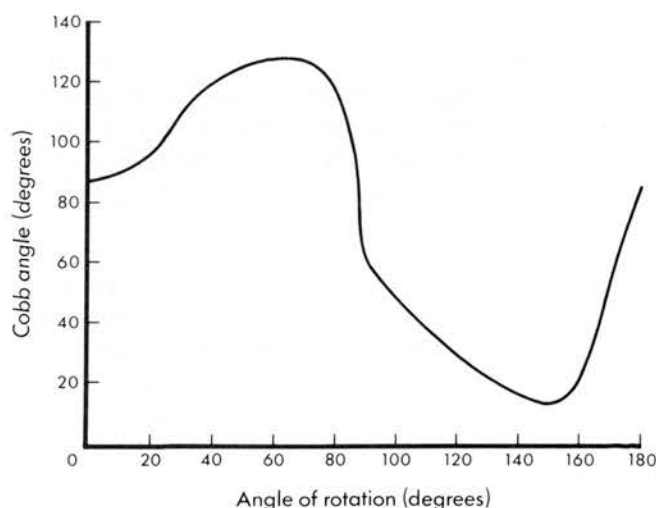


Fig. 1

Change in the measured Cobb angle with rotation (Specimen 1).

in rotation from the true anteroposterior view. This represents a true lateral projection of the apex of the deformity and if measurements of the curve at this point are restricted to the truly lateral apical vertebrae then a mean lordosis of 14° is observed. With further rotation the apparent Cobb angle of the deformity increases again until it reaches its original value when the specimen has been rotated through 180°.

Figures 2 to 5 show sample radiographs obtained at four points during the rotational cycle: an anteroposterior view of the specimen, an anteroposterior view of the deformity, a lateral view of the specimen, and a lateral view of the deformity. The anteroposterior and lateral views of the specimen itself are entirely misleading; they do not reflect the true magnitude of the deformity and merely represent two oblique views. The anteroposterior view of the specimen underestimates the deformity while the lateral view creates the erroneous impression of the presence of a kyphosis. Inspection of the lateral view of the apex confirms that there is no kyphosis; on the contrary there is a lordosis.

Table I summarises the radiographic data obtained on the 11 specimens. It can be seen that in all except the mildest curve (Specimen 3) the anteroposterior Cobb angle of the specimen underestimated the true anteroposterior magnitude of the deformity considerably. Furthermore, the lateral projection of the spine in all but one case (the smallest scoliosis with no rotational component) produced the spurious impression of a kyphosis while a true lateral projection of the apex of the curve revealed the important lordosis.

There was a significant correlation ($P < 0.01$) between the true extent of the deformity in the 11 specimens



Fig. 2



Fig. 3



Fig. 4



Fig. 5

Radiographs obtained at four points during the rotational cycle of an articulated scoliotic spine (Specimen 1). Figure 2—An anteroposterior view of the specimen with an apparent Cobb angle of 87°. Figure 3—Anteroposterior view of the scoliosis with a true Cobb angle of 128°. Figure 4—Lateral view of the specimen with an apparent kyphosis of 61°. Figure 5—Lateral view of the scoliosis with a true apical lordosis of 14°.

Table I. Radiographic measurements (in degrees) of the 11 specimens

	Specimens											Mean
	1	2	3	4	5	6	7	8	9	10	11	
Cobb angle in AP view of specimen	87	21	18	43	41	53	104	122	75	103	105	70
Maximum Cobb angle in true AP view of deformity	128	40	18	64	55	58	125	184	114	135	174	99
Rotation (0 to max.)	65	50	0	50	50	25	35	70	65	60	90	50
Lateral profile of specimen	K61	K15	L3	K37	K23	K15	K16	K90	K31	K75	K94	K41
Lateral profile of curve apex	L14	L10	L3	L9	L8	0	L18	L19	L25	L22	L32	L14

K, kyphosis
L, lordosis

and the amount of axial rotation at the apex, showing that, as the lordosis rotates further and further to the side, it produces a bigger and bigger anteroposterior deformity. Furthermore, there was a significant correlation ($P < 0.01$) between the true size of the deformity and the amount of lordosis; the bigger the lordosis the bigger the secondary deformity it produced.

Morphometric analysis. All the spinal columns were longer anteriorly than posteriorly, that is, the mean anterior length (46.1 cm) was significantly greater than the mean posterior length (40.7 cm; $P < 0.01$). Table II shows the anterior and posterior heights of the vertebrae in the structural curve of each of the seven specimens measured; it shows that while the vertebrae above and

below the region of the curve apex retain their normal kyphotic configuration, posterior height being greater than anterior, a reversal occurs at the curve apex where the specimens were truly lordotic at bony level, the anterior vertebral height being greater than the posterior ($P < 0.02$).

DISCUSSION

Although there is considerable merit in trying to quantify a complex three-dimensional deformity as simply as possible (Ferguson 1930; Cobb 1948; Nash and Moe 1969), it is abundantly clear from these results that conventional methods of measuring the deformity in idiopathic scoliosis depend entirely on the radiographic projection.

Table II. Anterior and posterior heights (in centimetres) of vertebrae in the structural curves

Vertebra	Specimens													
	1		2		3		4		5		6		7	
	A	P	A	P	A	P	A	P	A	P	A	P	A	P
T1			1.32	1.35										
T2			1.73	1.76	1.68	1.63								
T3			1.78	1.56	1.63	1.60								
T4			1.72	1.54	1.63	1.61							1.68	1.74
T5			1.60	1.61	1.64	1.50					1.82	1.85	1.82	1.84
T6			1.83	1.97	1.69	1.67			1.95	2.15	1.81	1.84	1.60	1.45
T7			1.64	1.92	1.70	1.70	1.77	1.82	1.88	2.19	1.59	1.88	1.85	1.25
T8	2.26	1.77	1.73	1.75	1.81	1.87	1.88	1.64	1.96	1.66	1.90	1.70	1.80	1.38
T9	2.25	1.45	1.95	1.97	1.82	1.87	2.04	1.52	2.23	1.81	1.91	1.54	2.15	1.49
T10	2.41	1.25	2.10	2.18			2.05	1.45	2.35	2.10	1.89	2.09	2.27	1.85
T11	2.45	1.45	2.22	2.32			2.20	2.06	2.05	2.26			2.36	2.46
T12	2.46	1.31					2.31	2.04					2.37	2.60
L1	3.24	2.25					2.36	2.45						
L2	3.28	2.59												

A, anterior; P, posterior
T, thoracic; L, lumbar

Indeed an anteroposterior projection of the patient is really only anteroposterior as regards the vertebrae above the scoliosis and those below, not for those in the structural curve itself (Fig. 2). It is the three-dimensional nature of the deformity which renders uniplanar measurements fallacious. This is exemplified by the fact that the true anteroposterior magnitude of the deformity was on average 41% greater than that obtained from the anteroposterior projection of the specimen; similarly the apparently kyphotic lateral profile varied by an average of 55° from the true lateral projection which showed the lordosis.

It is important to realise that curve size and rotation are significantly related. An anteroposterior view of the patient underestimates the true extent of the deformity, and this phenomenon becomes more marked with increasing curve size. As the secondary deformity rotates past the coronal plane so it is directed progressively more posteriorly; thus an anteroposterior view of the patient will demonstrate a reducing Cobb angle when in fact quite the reverse is happening to the deformity. This trend continues until a plane is reached where there is little or no deformity on an anteroposterior view of the patient while a view at 90° to this will demonstrate the full deformity *en face*. This explains why surgeons may

be under the misapprehension that a serious deformity is improving when the opposite is in fact occurring.

True lateral projections of the apex of the curve demonstrate the presence of a lordosis; its fundamental role in progression of the deformity is supported by its close correlation with the size of the scoliosis and with the amount of axial rotation. Although overall morphometric measurements of the spinal columns demonstrate that anterior height is excessive, this is particularly significant at the apical segments of the structural curves where a lordosis exists at bony level. These measurements confirm that in idiopathic scoliosis the apical vertebral bodies are wedge-shaped in the opposite direction to that in Scheuermann's disease.

True measurement of the size of the deformity is an important consideration in investigations of the natural history and treatment of idiopathic scoliosis. This study demonstrates the three-dimensional nature of the deformity in scoliosis and its property of changing in character and magnitude according to the vertebral level under consideration, and the plane of radiographic projection. It is unique as it is not ethically possible to obtain multiple radiographs, taken in small serial increments of rotation, of the deformed spines of otherwise normal children. The specimens studied are admirably suitable for this purpose.

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REFERENCES

- Adams W. *Lectures on the pathology and treatment of lateral and other forms of curvature of the spine*. London: John Churchill and Sons, 1865.
- Cobb JR. Outline for the study of scoliosis. *Am Acad Orthop Surg Inst Course Lect* 1948;5:261-75.
- Dickson RA, Lawton JO, Archer IA, Butt WP. The pathogenesis of idiopathic scoliosis: biplanar spinal asymmetry. *J Bone Joint Surg [Br]* 1984;66-B:8-15.
- du Peloux J, Fauchet R, Faucon B, Stagnara P. Le plan d'élection pour l'examen radiologique des cypho-scolioses. *Rev Chir Orthop* 1965;51:517-24.
- Ferguson AB. The study and treatment of scoliosis. *South Med J* 1930;23:116-120.
- Nash CL Jr, Moe JH. A study of vertebral rotation. *J Bone Joint Surg [Am]* 1969;51-A:223-9.
- Whittle MW, Evans M. Instrument for measuring the Cobb angle in scoliosis. *Lancet* 1979;i:414.

COMBINED IDIOPATHIC KYPHOSIS AND SCOLIOSIS. AN ANALYSIS
OF THE LATERAL SPINAL CURVATURES ASSOCIATED WITH
SCHEUERMANN'S DISEASE.

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If idiopathic thoracic scoliosis and Scheuermann's disease are the opposite conditions biologically as well as biomechanically then the deformities cannot co-exist at the same site. This analysis of 50 patients with Scheuermann's kyphosis demonstrated that 70% had a lateral curvature of the spine. A minority were mild and at the site of the kyphosis indicating that the kyphotic process was slightly asymmetrical in the coronal plane. These lateral curvatures were not rotated. By contrast 30 patients with Scheuermann's disease had an idiopathic scoliosis either above or more usually below separated from the kyphosis by four or five spinal segments. These lordo-scolioses occurred where the spine is compensatorily hyperlordotic to balance the area of Scheuermann's disease. The study strongly indicated that idiopathic scoliosis and Scheuermann's disease share a similar pathological process affecting the vertebral end plate.

COMBINED IDIOPATHIC KYPHOSIS AND SCOLIOSIS

AN ANALYSIS OF THE LATERAL SPINAL CURVATURES ASSOCIATED WITH SCHEUERMANN'S DISEASE

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A radiological study of 50 patients with thoracic Scheuermann's disease revealed two types of lateral spinal curvature. A total of 43 lateral curves was present in 35 of the patients. Thirteen were apical at the same level as the Scheuermann's kyphosis and were due to vertebral-body wedging in the coronal plane; these curves had a mean Cobb angle of 15° , occurred with equal prevalence in boys and girls and were directed equally to right and left. Thirty curves occurred in regions of compensatory lordosis (mean 5.6°) situated above or, more commonly, below the Scheuermann's kyphosis. These scolioses had a mean Cobb angle of 16° , were more often convex to the right than to the left and were significantly more prevalent in girls than in boys.

The presence of these kyphoses and scolioses in the same spine, separated by only a few vertebrae, emphasises the importance of the sagittal plane in idiopathic spinal deformities and strongly suggests that idiopathic scoliosis and Scheuermann's disease share a common pathological process.

In both idiopathic kyphosis (Scheuermann's disease) and idiopathic scoliosis there is an abnormality of vertebral shape in the median sagittal plane. In Scheuermann's disease the apical vertebral bodies are wedged, with posterior height greater than anterior height, producing the kyphotic deformity (Scheuermann 1920). This is a rotationally stable configuration, since the axis of rotation of a kyphosis is situated anteriorly and the deformity therefore exists largely in one plane only. In idiopathic scoliosis, however, the vertebral bodies are wedged in the opposite direction, with anterior height greater than posterior height, so that there is a lordosis at the apex of the curve (Dickson *et al.* 1984; Deacon, Flood and Dickson 1984). This is a rotationally unstable situation and the lordosis readily rotates to one side, producing a scoliosis as a secondary deformity in the coronal plane.

Scheuermann's disease and idiopathic scoliosis both occur in otherwise normal growing children. They have similar prevalence rates and both exhibit a familial tendency (Sorenson 1964; Wynne-Davies 1968; Dickson 1983). It has been reported that a mild scoliosis is not

uncommon in patients with Scheuermann's disease (Sorenson 1964; Bradford, Moe and Winter 1975) although the characteristics of these scolioses have not been accurately determined. Since these two idiopathic deformities have similar epidemiological features and share the common denominator of sagittal plane asymmetry, we decided to study the lateral spinal curvatures associated with Scheuermann's disease.

PATIENTS AND METHODS

Fifty patients with thoracic Scheuermann's disease who were attending the scoliosis clinics at Leeds and Hull were studied. All the patients satisfied both clinical and radiological criteria for the diagnosis of Scheuermann's disease—that is, there was a fixed increase in the normal thoracic kyphosis, with vertebral-body wedging of 5° or more over three consecutive levels on lateral radiographs (Sorenson 1964). None of the patients had any significant leg-length inequality. There were 22 boys and 28 girls and their ages ranged from 10.4 to 19.1 years, with a mean of 14.9 years (girls 14.3 years, boys 15.4 years). Anteroposterior and lateral radiographs of the thoracic and lumbar spines were obtained in the erect position in all cases. The spinal curves in both coronal and sagittal planes were measured and recorded using Whittle's adaptation of Cobb's method (Whittle and Evans 1979). In addition, for each curve the apical and end-vertebrae were recorded. The presence and direction of rotation also were noted. Statistical significance was determined using the chi-squared and Fisher's exact probability tests.

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RESULTS

Details of the Scheuermann's deformity in the 50 patients are shown in Table I. The size of the kyphosis was similar for both boys and girls, with a similar number of vertebrae being wedged to the same extent. Lateral spinal curves were present in 35 (70%) of the patients. There was no significant correlation between the presence or size of a lateral curve and the overall size of the Scheuermann's kyphosis or the severity of vertebral wedging in the sagittal plane ($P < 0.05$). There were 43 lateral curves in all and these were of two types.

Thirteen of the lateral curves had their apex at the same level as that of the Scheuermann's kyphosis; their characteristics are detailed in Table II. They occurred with equal prevalence in both sexes and their convexities were directed almost equally to right and left. These lateral curves were due to asymmetry of the kyphosis with vertebral-body wedging in both coronal and sagittal planes. Few of these curves had any rotation and where rotation was present it was such that the spinous processes were directed towards the curve convexity, indicating that it was a kyphotic region of the spine which had rotated.

Thirty of the lateral curves were situated either above or below the apex of the kyphosis; their characteristics are detailed in Table III. They were significantly more common in girls than in boys ($P < 0.02$), though there was no significant difference between the sexes in either the size or the direction of the curve ($P > 0.05$). All these scolioses were associated with rotation; the spinous processes were directed towards the concavity of the curve, indicating that they were occurring in regions of lordosis which had rotated. This was confirmed when their lateral profiles were examined. Measurements in the sagittal plane of the apical three vertebrae in the scolioses revealed a mean lordosis of 5.6° .

Fifteen of the scolioses were apical in the thoracic spine and these showed the greatest right-sided predominance (11R, 4L); nine were thoracolumbar (5R, 4L) and six were apical in the lumbar region (3R, 3L). Twenty-three were below the Scheuermann's kyphosis and seven above. In eight cases there was an asymmetric kyphosis in association with a scoliosis, though 22 of the scolioses were adjacent to kyphotic deformities with no lateral deviation. Nineteen scolioses were greater than 10° in size (Table III) and these were commoner in girls and were significantly more often convex to the right than to the left ($P = 0.05$).

DISCUSSION

These measurements characterise the coronal plane curves associated with Scheuermann's disease and demonstrate that while a lateral curve in the form of an asymmetric kyphosis, due to vertebral-body wedging in both coronal and sagittal planes, is not unusual (Fig. 1), a true idiopathic scoliosis is more common (Figs 2 and 3). These idiopathic scolioses occur in regions of

Table I. Details of kyphoses (giving the mean and standard deviation or range)

	Number	Per-centage	Kyphosis angle (degrees)	Number of vertebrae	Wedging of vertebrae (degrees)	Thoracic kyphosis (degrees)
Boys	22	44	33 ± 13	4 (3-7)	8.2	45 ± 12
Girls	28	56	35 ± 13	4 (3-8)	8.8	50 ± 13
Whole group	50	100	34 ± 13	4 (3-8)	8.5	48 ± 13

Table II. Details of the lateral curvatures associated with the kyphosis

	Number	Per-centage	Cobb angle (degrees)		Curve convexity	
			Mean	Range	Left	Right
Boys	6	27	14.5	6-30	2	4
Girls	7	25	14.7	4-32	4	3
Whole group	13	26	14.6	4-32	6	7

Table III. Details of scolioses above or below the kyphosis

		Per-centage	Cobb angle (degrees)		Curve convexity	
Number			Mean	Range	Right	Left
<i>All magnitudes</i>						
Boys	9	41	14.1	8-30	6	3
Girls	21	75	16.4	5-32	13	8
Whole group	30	60	15.7	5-32	19	11
<i>Scoliosis > 10° only</i>						
Boys	5	23	18.2	12-30	5	0
Girls	14	50	20.6	12-32	10	4
Whole group	19	38	19.9	12-32	15	4

rotationally unstable lordosis above or below the Scheuermann's kyphosis and are therefore true lordoscolioses. These lordoscolioses have the typical right-sided and female predominance characteristic of adolescent idiopathic scoliosis and, as might be expected, these features become more evident with increasing size of the curve. Measurements of the lateral profiles of their apices confirmed the presence of a lordosis, as is always the case in idiopathic scoliosis.

Although these measurements were made on lateral radiographs of the patients and not true laterals of the rotated apices of the scolioses, and therefore undoubtedly underestimate the true size of the apical lordosis, the error introduced is only slight, since the curves were mostly of small magnitude with only small amounts of rotation. Indeed, the scolioses associated with Scheuermann's disease seldom progress to such a size



Fig. 1

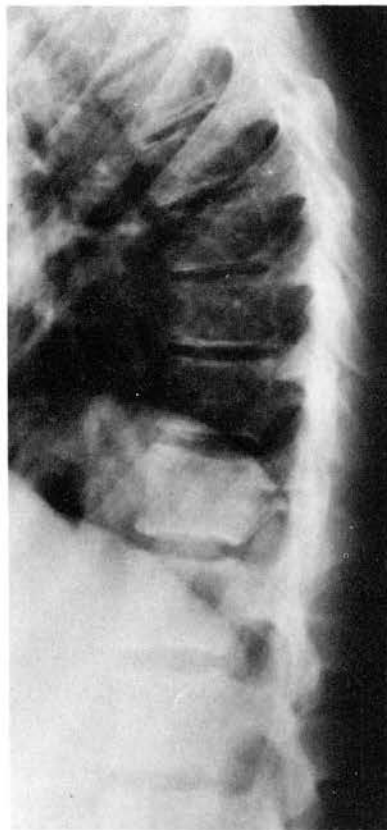


Fig. 2



Fig. 3

Figure 1—Anteroposterior radiograph showing a mild lateral curvature; this is at the level of a Scheuermann's kyphosis and is due to vertebral wedging in the coronal plane. Note the absence of rotation. Figure 2—Lateral radiograph showing Scheuermann's kyphosis in the mid-thoracic region with a compensatory lordosis below. Figure 3—Anteroposterior radiograph of the same patient showing scoliosis below the region of kyphosis. Note the rotation with the posterior elements directed towards the concavity.

that surgical correction is required, since the adjacent region of increased kyphosis maintains the axis of spinal rotation relatively anteriorly and thus protects against rotation. In addition, and of more importance, the mean age of onset of Scheuermann's disease is late in the adolescent period and this limits the potential for the scolioses to progress with growth. A further consequence of the rotational stability of kyphoses is that the Scheuermann's deformity remains in the sagittal plane and vertebral wedging proceeds inexorably with growth, so that a severe kyphosis can result. In contradistinction, the lordosis of idiopathic scoliosis is rotationally unstable and moves progressively further from the sagittal plane, giving relief of compression on the posterior regions of the growth plates and the amount of vertebral wedging is less marked (Fig. 4).

The characteristics of the Scheuermann's kyphosis and the degree of vertebral-body wedging in these patients are similar to those of previously reported series. Although a review of the literature (Sørensen 1964) would seem to suggest an equal sex incidence, our group contained a greater number of girls than boys as did the more recent series. This may indicate that girls complain of kyphosis more readily than boys—perhaps because in most cases Scheuermann's disease is merely a cosmetic deformity (Kling and Hensinger 1984).



Fig. 4

Lateral radiograph of patient with adolescent idiopathic scoliosis. Note the lordosis due to vertebral-body wedging posteriorly with end-plate changes similar to those seen in Scheuermann's disease.

Although both Scheuermann's disease and idiopathic scoliosis are due to asymmetry in the sagittal plane, the exact nature of the underlying pathological changes producing these deformities remains obscure. Widely regarded as being multifactorial in origin, it is clear that the lesion must lie in the vertebral growth plate. This study demonstrates that it is not unusual for closely adjacent vertebrae to exhibit wedging either anteriorly, posteriorly or laterally, thus producing deformities in both the sagittal and coronal planes. That these kyphoses and scolioses occur in the same spine with their apices separated by only a few vertebral levels confirms the importance of asymmetry of vertebral shape in the sagittal plane in the production of idiopathic spinal deformities and strongly suggests a common underlying pathological process. It is not necessary, however, to consider this process as truly pathological; it may merely be secondary to the biomechanical consequences of sagittal spinal shape at opposite extremes of a normal spectrum occurring in adolescence (Willner 1981).

REFERENCES

- Bradford DS, Moe JH, Winter RB. Scheuermann's juvenile kyphosis and postural round back deformity. In: Rothman RH, Simeone FA, eds. *The Spine*. Philadelphia: WB Saunders, 1975;1:361-79.
- Deacon P, Flood BM, Dickson RA. Idiopathic scoliosis in three dimensions: a radiographic and morphometric analysis. *J Bone Joint Surg [Br]* 1984;66-B:509-12.
- Dickson RA. Scoliosis in the community. *Br Med J* 1983;286:615-8.
- Dickson RA, Lawton JO, Archer IA, Butt WP. The pathogenesis of idiopathic scoliosis: biplanar spinal asymmetry. *J Bone Joint Surg [Br]* 1984;66-B:8-15.
- Kling TF Jr, Hensinger RN. Scheuermann's disease: natural history, current concepts and management. In: Dickson RA, Bradford DS, eds. *Management of spinal deformities*. (Butterworths International Medical Reviews: Orthopaedics 2). London: Butterworths, 1984.
- Scheuermann HW. Kyfosis dorsalis juvenilis. *Ugeskr Laeger* 1920;82:384-93.
- Sørensen KH. *Scheuermann's juvenile kyphosis: clinical appearances, radiography, aetiology and prognosis*. Copenhagen: Munksgaard, 1964.
- Whittle MW, Evans M. Instrument for measuring the Cobb angle in scoliosis. *Lancet* 1979;i:414.
- Willner S. Spinal pantograph: a non-invasive technique for describing kyphosis and lordosis in the thoraco-lumbar spine. *Acta Orthop Scand* 1981;52:525-9.
- Wynne-Davies R. Familial (idiopathic) scoliosis: a family survey. *J Bone Joint Surg [Br]* 1968;50-B:24-30.

AETIOLOGY OF IDIOPATHIC SPINAL DEFORMITIES.

Dickson RA.

Archives of Disease in Childhood; 60: 508-511, 1985.

This paper further explored lateral spinal profile indicating that at the ranges of normality of lateral spinal shape were children at one end who could be defined as Scheuermann's disease and those at the other end who could be defined as having idiopathic lordo-scoliosis such that both deformities could be explained without any specific pathological process being present.

Aetiology of idiopathic spinal deformities

R A DICKSON

Aetiology of idiopathic spinal deformities

There are two types of idiopathic spinal deformity idiopathic scoliosis, and idiopathic kyphosis (Scheuermann's disease). Both conditions affect otherwise entirely normal, healthy children and while idiopathic scoliosis has two peaks of incidence during infancy and early adolescence, idiopathic kyphosis is mainly prevalent in late adolescence. Idiopathic scoliosis and kyphosis share many features, being modal at the same site with a common apex at the eighth and ninth thoracic vertebral bodies and having a similar familial trend and community prevalence rate. As will be seen, they also share a common 'pathological process'.

Idiopathic scoliosis

Because many disorders of nerve or muscle are associated with a scoliosis most workers attempting to discover the cause of idiopathic scoliosis have focussed on neuromuscular factors. No such factors, or any other structural or functional abnormalities, have been found which are not secondary to the presence of a spinal deformity.¹ Moreover, with two per cent of teenagers showing an idiopathic scoliosis of 10° or more, a hitherto unknown neuromuscular disorder of epidemic proportions has to be incriminated. What has not received sufficient attention is the nature of the idiopathic scoliotic deformity.

Three dimensional deformity. A postero-anterior radiograph of the spine of a patient with idiopathic thoracic scoliosis (Fig. 1) shows the three components of the deformity, one of which is aetiologically crucial. The lateral curvature of the spine with rotation of the vertebrae within the curve are the two most obvious, most described, but least important features. Wherever the idiopathic scoliotic deformity occurs in the spine, for example thoracic, thoraco-lumbar, or lumbar, the direction of rotation is the same, such that the posterior elements rotate into the curve concavity while the vertebral bodies rotate anteriorly into the curve convexity. If a line, imaginary or true, is now drawn down the tips of the spinous processes throughout the curve and a similar line is drawn through the centre of the vertebral bodies, it will be seen that the line joining the spinous processes is the shorter. Therefore, the back of the spine is shorter than the front and the entire deformity must be lordotic.²



Fig. 1 Postero-anterior radiograph of a patient with idiopathic thoracic scoliosis. There is a lateral curvature of the spine with rotation such that the spinous processes are directed towards the curve concavity.

This elementary but important geometrical point seems to have received little emphasis, and, additionally, spurious impressions of the three dimensional nature of the idiopathic scoliotic deformity have been perpetuated by the taking of inappropriate radiographic views. Because of the rotational component a posterior-anterior radiograph of the patient must, by definition, be an oblique view of the deformity and so, of course, is a lateral view of the patient. In order to take true planar views either the beam or patient must be rotated according to the amount of rotation of that particular vertebra, and when a true lateral projection of the curve apex is obtained (Fig. 2) the lordosis is clearly visualised.³ It is attributable solely to sagittal spine shape, the anterior height of the vertebral bodies being greater than the posterior and any Schmorl node

formation or end plate irregularity more posteriorly. Disc height remains symmetrical and does not contribute to the lordosis.

Normal sagittal spine shape. The presence of a lordosis in the thoracic region implies a radical alteration in sagittal spine shape. There is normally a kyphosis here, although the spine above and below is naturally lordotic. The axis of spinal rotation runs through these sagittal spine curves so that the normal thoracic kyphosis, lying behind the axis of rotation and therefore being under tension and not compression, is protected from rotation. In the presence of a thoracic lordosis the axis of spinal rotation will run behind the front of the vertebral bodies, which, particularly on forward flexion, undergo compression and must rotate to the side to



Fig. 2 (left) True lateral radiograph of the apical region of an idiopathic thoracic scoliosis. There is a lordosis at bony level.

(above) Lateral radiograph of the thoracic spine of a patient with Scheuermann's disease. There is a kyphosis at bony level.

be accommodated (the rotational prominence in idiopathic scoliosis is much more noticeable on forward flexion).

In the cervical and lumbar regions lordoses are normal but they are protected from rotational instability by having a large amount of segmental movement available, by the presence of powerful muscles and fascial systems behind them, and by squat vertebrae with a broad front which tend to resist rotation. Moreover, these are at the extreme ends of the spine which naturally tend to remain facing the front. Idiopathic scoliosis may occur, however, if the lumbar lordosis is increased or if stiffness is superimposed. Thus, while the term 'kypho-scoliosis' is what most practitioners recall of spinal deformities, it is a combination which cannot and does not exist.²

Idiopathic kyphosis (Scheuermann's disease)

The lateral radiograph of the thoracic spine in Scheuermann's disease (Fig. 2) illustrates the same vertebral changes as in idiopathic scoliosis, but in the opposite direction with much reduced anterior vertebral height compared with posterior, and Schmorl node formation and end plate irregularity being situated anteriorly.⁴ Again these are otherwise entirely healthy normal children. If the process of vertebral wedging is asymmetric (for example the right side of the vertebra being more affected than the left) then a mild scoliosis can exist at the same site as the kyphosis but with the spinous processes rotated towards the curve convexity, the opposite direction to that occurring in idiopathic scoliosis, as would be entirely expected with an asymmetric kyphosis. The anteriorly situated axis of spinal rotation ensures, however, that the kyphotic region itself does not rotate. Where a severe scoliosis can occur in the spine of a patient with Scheuermann's disease, is below the area of kyphosis, where there is a compensatory lordosis, which as in idiopathic scoliosis is vulnerable to rotation. Therefore in more than 50% of patients with Scheuermann's disease there is a true idiopathic scoliotic deformity apical some five vertebrae below the apex of the kyphosis. Thus in these patients the same otherwise normal child has the deformities of both Scheuermann's disease and idiopathic scoliosis but not at the same site. This lends strong support to a common pathological process.

Normal lateral spinal profile

The normal thoracic kyphosis extends from the third to the tenth thoracic vertebrae, those above belong-

ing to the cervical lordosis and those below belonging to the lumbar lordosis. During late childhood and early adolescence, between the ages of 8 and 12 years, the normal thoracic kyphosis reduces in size and this occurs at the same time in both boys and girls.⁵ Thus, during this period, those with an excessive amount of flattening become truly lordotic with obvious rotational consequences, and in girls this process is occurring during the phase of increased growth velocity of early adolescence. This explains why girls are particularly vulnerable to the deformity of idiopathic scoliosis. By the time boys go through their peak adolescent growth velocity the thoracic kyphosis has become fully re-established: they are relatively protected from idiopathic scoliosis, therefore, but are particularly prone to the opposite deformity of Scheuermann's disease, which is not prevalent until later adolescence. Lateral profile is governed, like other aspects of body shape, genetically, and this explains the familial trend observed in both idiopathic scoliosis and Scheuermann's disease.⁶ The scoliotic patient, however, with a flattened lateral profile seems marginally taller than a peer, without there being any other growth abnormality, because of the uncoiling effect.

Treatment considerations

The kyphotic spine clearly requires extension so that the anterior aspects of the growth plates can be unloaded, and if a brace or cast which maintains spinal extension is applied then there is true physiological correction of sagittal spinal shape. Scheuermann's disease, therefore, is eminently treatable conservatively. The thoracic lordosis of idiopathic scoliosis would, however, require flexion and this is precisely when the deformity is rotationally unstable. Accordingly, the deformity of idiopathic scoliosis is not treatable conservatively² and, while it can be envisaged that the wearing of a brace 23 hours a day will at least prevent the patient from the unfavourable consequences of leaning forward, there is no clear evidence that it alters the natural history of the condition.⁷ The deformity of idiopathic scoliosis can, therefore, only be corrected surgically. Harrington instrumentation, which is attached above and below the area of rotational deformity, cannot be expected to alter the rotational prominence with which every teenager presents and this is borne out by studies using computed tomography.⁸ Only by altering the shape of the spine in the sagittal plane can the normal thoracic kyphosis be restored, the spine derotated, all three planes of the deformity thereby corrected, and the patient's presenting complaint effectively dealt with.⁴

References

- ¹ Zetterberg C, Aniansson A, Grimby G. Morphology of the paravertebral muscles in adolescent idiopathic scoliosis. *Spine* 1983;**8**:457-62.
- ² Dickson RA, Lawton JO, Archer IA, Butt WP. The pathogenesis of idiopathic scoliosis. *J Bone Joint Surg* 1984;**66B**:8-15.
- ³ Deacon P, Flood BM, Dickson RA. Idiopathic scoliosis in three dimensions. A radiographic and morphometric analysis. *J Bone Joint Surg* 1984;**66B**:509-12.
- ⁴ Dickson RA, Lawton JO, Butt WP. The pathogenesis of idiopathic scoliosis. In: Dickson RA, Bradford DS, eds. *Management of spinal deformities*. London: Butterworths, 1984:1-37.
- ⁵ Willner S. Spinal pantograph—a non-invasive technique for describing kyphosis and lordosis in the thoraco-lumbar spine. *Acta Orthop Scand* 1981;**52**:525-9.
- ⁶ Wynne-Davies R. Familial (idiopathic) scoliosis. A family survey. *J Bone Joint Surg* 1968;**50B**:24-30.
- ⁷ Kaiser RP, Shufflebarger HL. The Milwaukee brace in idiopathic scoliosis. Evaluation of 123 completed cases. *Clin Orthop* 1976;**118**:19-24.
- ⁸ Aaro S, Dahlborn M. The effect of Harrington instrumentation on the longitudinal axis rotation of the apical vertebra and on the spinal and rib-cage deformity in idiopathic scoliosis studied by computer tomography. *Spine* 1982;**7**:457-62.

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THE PATHOGENESIS OF IDIOPATHIC SCOLIOSIS.

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In 'Scoliosis Prevention' (Warner JO and Mehta MH, Eds) Praeger:
New York etc, 64-79, 1985.

This chapter restated many of the above pathogenetic observations and discussed them in relationship to the clinical features of idiopathic scoliosis with particular reference to recreation of the thoracic kyphosis so that growth and mechanics would be harnessed in favour of the patient thus mitigating any future likelihood of curve deterioration after surgical intervention.

5

THE PATHOGENESIS OF IDIOPATHIC SCOLIOSIS

R.A. Dickson, J.O. Lawton, P. Deacon, I.A. Archer,
and B.M. Flood

INTRODUCTION

At the 1979 counterpart of this symposium, Professor Nachemson, having perused the world literature on the causes of idiopathic scoliosis, enumerated 22 significant pathological findings that had been reported in patients with idiopathic scoliosis (1). Humorously, but seriously, he posed the question, "Is it really possible that our clinically healthy scoliotic patients can have all these abnormalities, or are we looking at the wrong things?" The Leeds Scoliosis Study Group would endorse this view and would add that such findings as may be unequivocal still put us some way from understanding the mechanism of production of the idiopathic scoliotic deformity. Accordingly, our approach has been rather different: We have started with an analysis of the deformity and worked from there. Professor Nachemson also reminded us not to compare apples with oranges and suggested that a reasonable starting point in future research might be an analysis of idiopathic thoracic curves apical around T8. We have followed his advice virtually to the letter. The results of our investigations indicate that the shape of the lateral profile of the spine provides the key to understanding the development of both idiopathic scoliosis and Scheuermann's disease, relates to the growth and genetic considerations known to be important, and helps to explain the natural history of idiopathic deformities and their response to treatment.

THE DEFORMITY OF IDIOPATHIC SCOLIOSIS

Clinical Evidence

In describing the deformity of idiopathic scoliosis, standard anatomical nomenclature will be used (2). The plane that divides the

body into front and back halves is the coronal plane, the plane that divides the body into right and left halves is the median (sagittal) or more simply the median plane, and the plane dividing the body into cross sections is the transverse plane. This nomenclature will be used henceforth. We can learn a lot about the deformity of idiopathic scoliosis when a PA radiograph of a patient is inspected (Figure 5.1). Interestingly, although this is the standard view for assessment and measurement purposes, it is in fact a PA of everything except the structural curve because of the concomitant rotation. Rotation always occurs in the same direction, no matter where the idiopathic deformity is in the spine. The vertebral bodies rotate toward the curve convexity, while the posterior elements are directed toward the curve concavity. If a normal lumbar lordosis rotates to the side it will produce a secondary deformity in the coronal plane with the posterior elements directed toward the curve concavity, but if the normal thoracic kyphosis were to rotate to the side then it would produce a similar coronal plane curvature but with the spinous processes rotated toward the curve convexity. It can therefore be said with some certainty that in idiopathic thoracic scoliosis there must be a lordosis at the curve apex.

The important observations of Adams (3), who stated more than 100 years ago in his classic monograph before radiographs were available that "lordosis plus rotation equals lateral flexion," have not received the attention they deserve. Indeed, that the essential primary lesion of idiopathic scoliosis exists in the median plane in the nature of a lordosis has received only sporadic support (4-6), notably from Somerville and Roaf, neither of whom could conceive of the deformity as occurring in any other way. It is, however, difficult to visualize the true deformity of idiopathic scoliosis if only standard radiographs, PA and lateral of patient and not deformity, are obtained. Moreover, as rotation increases from the neutral vertebrae to the apex of the curve, it is not possible to obtain true PA or lateral radiographs of the entire deformity, only the individual vertebrae within it.

It is not always easy to see the apical lordosis in the thoracic region on inspection of the patient's back, although the lordosis is more easily seen when the scoliosis is small (Figure 5.2). There are two principal reasons for this. When the back of a patient is inspected, what are being observed are the posterior elements and not the vertebral bodies situated more anteriorly where the deformity really exists. Furthermore, the shortest distance down a structural curve is down the back (7,8), and therefore the posterior elements tend to remain very much straighter than the vertebral bodies in front of them. With bigger curves the apical lordosis is even less easily visible because if the deformity progresses the apex is directed



Figure 5.1 PA radiograph of an idiopathic thoracic scoliosis. The posterior elements are directed toward the curve concavity; a lordosis must be present.

(For a better quality picture see Publication 25, Figure 1.)

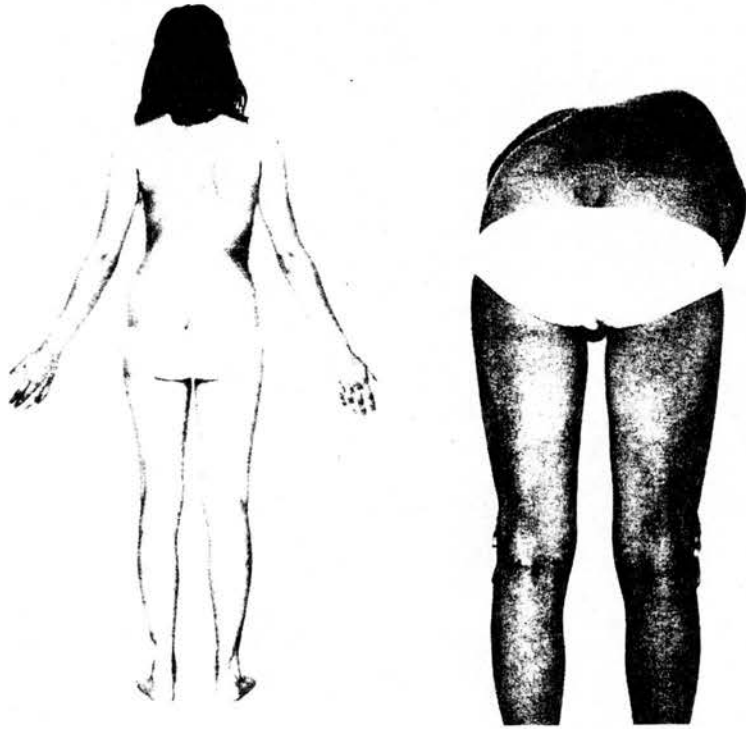


Figure 5.2 (a) Girl with idiopathic thoracic scoliosis in the erect position. There is a lordosis at the curve apex, and the rotational prominence is small. (b) On forward flexion the rotational prominence is maximal—the effect of a lordosis under compression.

not only laterally but also posteriorly and, compounded by the deceptive rib hump, gives the erroneous impression that a kyphoscoliosis exists when quite the reverse is the case.

An integral part of clinical examination, and the basis of the one-minute school screening test, is forward bending. In the erect position the appearance of the back is much less remarkable, while on increasing forward flexion the rotational prominence becomes progressively more obvious (Figure 5.2b). It is not difficult to envisage that this phenomenon may be due to the lordosis undergoing increasing compression on forward flexion such that it is compelled to rotate to the side in order to be accommodated.

The lateral profile of the spine would therefore appear to be particularly important, and this we have studied in some detail (8-12).

The Median Plane in Early Curves

The mean PA Cobb angle (PA of patient, not deformity) of the last 50 patients presenting to the Leeds Scoliosis Centre with late-onset idiopathic scoliosis was 40° . We thought it important to study the median plane in these patients before treatment of any kind could have influenced spinal shape. Lateral projections of the patient, like PA projections of the patient, must of necessity be oblique projections of the deformity, and therefore true lateral projections of the deformity (beam directed down rotational prominence) were obtained. Spinal



Figure 5.3 True lateral radiograph of the apex of an idiopathic thoracic curve revealing the important lordosis.

(For a better quality picture see Publication 11, Figure 2.)

shape in the median plane was measured by Whittle's adaptation of Cobb's method (13) and the mean overall thoracic kyphosis measured 20°, but at the curve apex, modal at D8, there was a lordosis of 5° on average, usually involving the three apical vertebrae (Figure 5.3). Significantly, this lordosis was at bony level, the intervertebral discs not contributing to the magnitude of the lordosis. Accordingly, the apical vertebrae were observed to be wedged, the anterior height being greater than the posterior height and any end-plate irregularity or Schmorl node formation situated more toward the posterior aspect of the vertebral growth plate (14).

The Median Plane in More Severe Curves

With more severe curves the illusion of a kyphoscoliosis is all the greater. With increasing rotation the apex of the deformity is directed more and more backward (indeed, this must be the case when a lordosis rotates). As the amount of apical rotation approaches 90°, a PA view of the patient appears more as a lateral view of the deformity, while a lateral view of the patient appears more as a PA view of the deformity (11). When there is 45° of apical rotation, such projections will produce two perfect oblique views of the apex. However, true lateral projections of the apices of severe deformities reveal even bigger lordoses, mean Cobb angle 14° in 11 articulated specimens so examined.

When such a specimen is rotated through 180° and X-rayed incrementally through that range, the kyphoscoliotic illusion is even better demonstrated (Figure 5.4). As the specimen is rotated from the PA of the specimen position, the Cobb angle of the deformity increases to a maximum and the apex of the deformity is truly seen in a PA projection, a view that has been referred to by Stagnara as the plan d'election (15). The graph demonstrates clearly that a PA and lateral projection of the specimen record two very similar deformities, 90° of rotation apart, on each side of the position of the plan d'election. More importantly, 90° further round than the plan d'election is where the deformity is seen in a true lateral projection. This is the projection that reveals flattening of the thoracic kyphosis and the apical lordosis. The size of the lordosis increases with the size of the scoliotic deformity, and all of 20° of lordosis can be observed in these severe deformities. Measurements of anterior and posterior vertebral body heights on true lateral projections again demonstrate that the lordosis exists at bony level.

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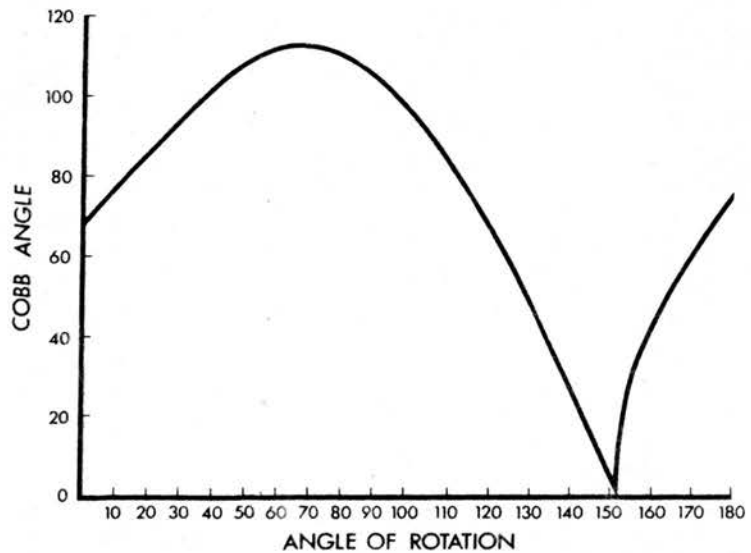


Figure 5.4 Graph illustrating the variation in curve size according to rotation of the X-ray beam. PA and lateral views of the patient are two oblique views of the scoliosis. True PA and lateral projections of the apex of the deformity reveal maximal curve size and a lordosis, respectively.

THE IMPORTANCE OF GROWTH

The importance of the phases of spinal growth as a critical environment during which idiopathic scoliosis commences and can progress, particularly in relation to the adolescent growth spurt, has long been known (16). Willner has studied growth extensively, and perhaps his most important contribution concerns the shape of the spine in the median plane and its alteration during the early phase of adolescent growth (17). He has shown that in both boys and girls the thoracic kyphosis diminishes considerably in the early adolescent phase. Significantly, this is occurring in girls during a period of rapidly increasing growth velocity, and it may account for the progression potential that characterizes the deformity in girls. In boys diminution of the thoracic kyphosis occurs during a period when growth velocity is constant, which may explain the relative protection from progression that boys enjoy. Interestingly, when the thoracic kyphosis in boys is maximal they are also going through peak adolescent growth velocity. This fact may explain why boys are particularly prone to develop the opposite deformity, Scheuermann's disease, in the median plane (14). Analysis of prevalence rates of idiopathic scoliosis with sex of the children also supports this concept,

with an even female-to-male sex ratio for small curves, rising to a great preponderance of females when the deformity achieves clinical significance (18).

THE BIOMECHANICS OF IDIOPATHIC SCOLIOSIS

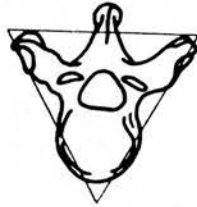
In the presence of a normal thoracic kyphosis the axis of spinal rotation lies anterior to the vertebral bodies and thus the kyphotic configuration protects against rotation. In the presence of an apical lordosis this axis is now displaced more posteriorly, such that the vertebral bodies, particularly on forward flexion, must rotate to the side in order to be accommodated (8). By contrast, conditions associated with an increased thoracic kyphosis do not have associated rotation, and the deformity therefore progresses essentially in one plane only.

If a thoracic lordosis were perfectly symmetrical, it could theoretically be in equilibrium even on forward flexion or would rotate to right or left at random. But that this does not occur strongly suggests the presence of spinal asymmetry in another plane, transverse, coronal, or both. The heart-shaped thoracic vertebral bodies, with a greater AP depth than lateral width, can be regarded as prisms with their apices anteriorly (Figure 5.5). Such a configuration demonstrates marked rotational instability on forward flexion as the apex of the prism moves to the side in order to counter compression. By contrast, the bodies of the cervical and lumbar vertebrae have a greater lateral width than AP depth and can be likened to prisms with their bases anteriorly, and this configuration is stable on forward flexion. Thus the apparent instability of the inbuilt cervical and lumbar lordoses is to a certain extent protected from rotation by the shape of their vertebral bodies.

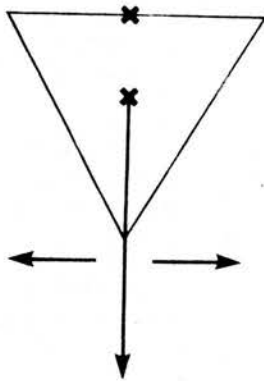
The thoracic prisms are, however, asymmetrical, being constantly flattened on the left side from T4 through T9 from the pressure effect of the descending thoracic aorta such that the apices of the prisms now lie to the right of the median plane. In the presence of a lordosis there will be a tendency for the prisms to rotate to the right.

Spinal shape in the coronal plane is also important, and epidemiological surveys indicate that, while curves of small magnitude (5° - 9°) have a left-sided preponderance, this changes with increasing curve magnitude such that deformities of clinical significance have a 10-to-1 right-to-left ratio (18). Left-sided curves would therefore tend to negate the tendency of the transverse plane to direct a curve to the right and may explain the relative protection from progression found with left-sided curves. The presence of a few degrees of

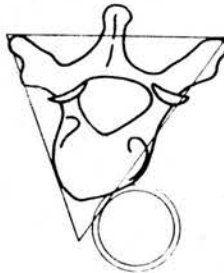
Figure 5.5 Thoracic vertebral shape.



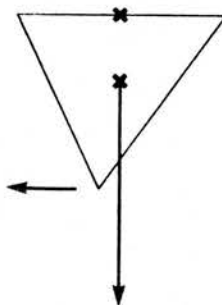
- (a) Thoracic vertebral bodies are shaped like prisms with the apex anteriorly.



- (b) A symmetrical prism could rotate to left or right in the presence of a lordosis.



- (c) The thoracic prisms are asymmetrical due to the aorta with the apex to the right of the median plane.



- (d) The prism will preferentially rotate to the right.

right-sided coronal plane asymmetry complements the transverse plane, and therefore more progression potential can be anticipated.

SCHEUERMANN'S DISEASE (IDIOPATHIC KYPHOSIS)

Scheuermann's disease would appear to be precisely the same deformity in the opposite direction in the median plane (8, 10, 11, 14). Lateral views of the apex of Scheuermann's deformity demonstrate reduced anterior vertebral height, and any end-plate irregularity or Schmorl node formation is situated more anteriorly (Figure 5.6). This point has already been observed by radiologists (14). Interestingly, Scheuermann's disease is also modal at D8 and has a not dissimilar prevalence rate to idiopathic scoliosis with evidence of a familial trend (19). Such a deformity, being a kyphosis, is protected from rotation.



Figure 5.6 Lateral radiograph of a Scheuermann's kyphosis. The deformity is the opposite of idiopathic scoliosis.

(For a better quality picture see Publication 11, Figure 3.)

It has been speculated that in one-third of cases of Scheuermann's disease there is a mild scoliosis present (19,20). We have studied the PA radiographs of patients with Scheuermann's disease and found that a scoliosis is in fact present in 80 percent of cases. In a minority, 15 percent of cases, the scoliosis is in the region of the Scheuermann's kyphosis and is in the nature of an asymmetric kyphosis with the spinous processes directed toward the curve convexity. Presumably the anterior wedging process has affected more the right or the left of the end-plate. In more than two-thirds of cases, however, there is true idiopathic scoliosis situated four or five segments above or below the apex of the kyphosis, and this is in the region of the compensatory lordosis, which has rotated to the side to produce the scoliotic appearance. The spinous processes in these deformities are directed toward the curve concavity. The average PA Cobb angle of these scolioses is only 15°, and the relatively benign course is related to a later age of onset of the deformity and an axis of spinal rotation that is not as far posterior as would be the case if the kyphosis were not present. We feel it is inconceivable that two similar deformities in opposite directions four or five vertebrae apart in the spines of otherwise entirely normal children can be anything else but the same "pathology."

THE IMPORTANCE OF STATURE

Several studies have suggested that girls with idiopathic scoliosis are taller than their peers (21-23), and one study using contemporaneous controls confirmed this but also demonstrated that they were no different from their peers with regard to any other variable concerning growth and maturity (9). We feel therefore that it is stature and not growth that is different and that the former can be simply explained on the basis that the normal spinal curvature in the thoracic region in the median plane is suppressed in idiopathic scoliosis. There is therefore an uncoiling effect in the median plane such that the patient will appear to be taller without there being any growth abnormality (Figure 5.7). This is supported by the observation from longitudinal studies that while such children are taller they are not growing faster (9).

The lateral profile of the spine in normal children has been studied, and three types of profile have been observed: flat backs, round backs, and those in between (24,25). In addition there would appear to be a familial trend for this appearance. This, we believe, is where genetic studies are relevant (26-29). The increased prevalence of idiopathic scoliosis in families does not, we think, reflect a gene for idiopathic scoliosis but indicates the genetically deter-

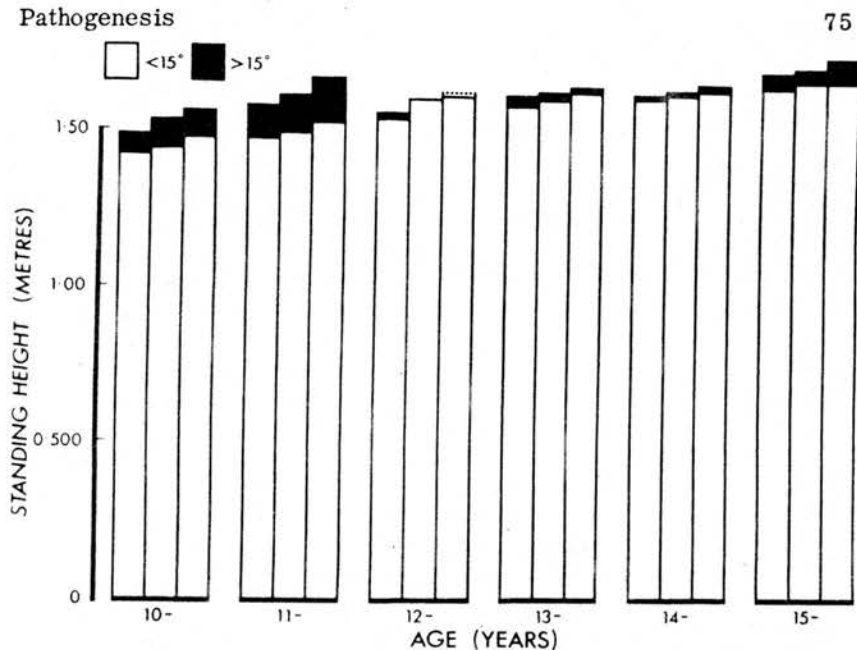


Figure 5.7 Histogram of mean standing heights of adolescent girls with idiopathic scoliosis. Those whose curves are 15° or more are significantly taller than both those with smaller curves and controls.

mined lateral profile, flat-backed children coming from flat-backed families with the danger of the rotational appearance of an idiopathic curve.

There would therefore appear to be a spectrum of lateral profiles, those with the flattest backs containing a lordosis producing idiopathic scoliosis on the one hand while at the other end of the spectrum those with the roundest backs have the clinical deformity of Scheuermann's disease. We do not therefore think it is necessary to contemplate a particular etiology or pathology for either idiopathic scoliosis or Scheuermann's disease if they are at opposite ends of a normal spectrum.

THE CLINICAL CONSEQUENCES OF SPINAL SHAPE IN THE MEDIAN PLANE

It would appear that the deformity of idiopathic scoliosis, a lordosis that has already rotated to the side, is virtually untreatable conservatively. What would be required is a method by which the thoracic kyphosis can be reconstituted, but any flexion of the lordo-scoliosis leads to further rotational progression. The best that

conservative treatment, brace or cast, can be expected to do is to prevent rotational progression by preventing forward spinal movement, and this statement is borne out by clinical experience (30). A partial correction of the scoliotic component can be produced by obliteration of the lumbar lordosis that gives rise in turn to thoracic extension, which puts the thoracic lordosis under less compression (31).

Roaf realized the need to correct the lordosis and favored anterior epiphysiodesis; but it was asking too much of growth to correct both elements of the deformity. The critically important advance of Luque in devising instrumentation (32) that can, in one procedure, reconstitute the thoracic kyphosis without the need for a stabilizing fusion would appear to be ideal for this purpose (Figure 5.8). Growth is harnessed in favor of the patient now that the mechanics have been corrected. We have necessarily been selective in using this approach, and our indications are that the deformity should have failed a conservative trial, it should be no greater than 50° (PA Cobb angle of patient), and there should be at least two years

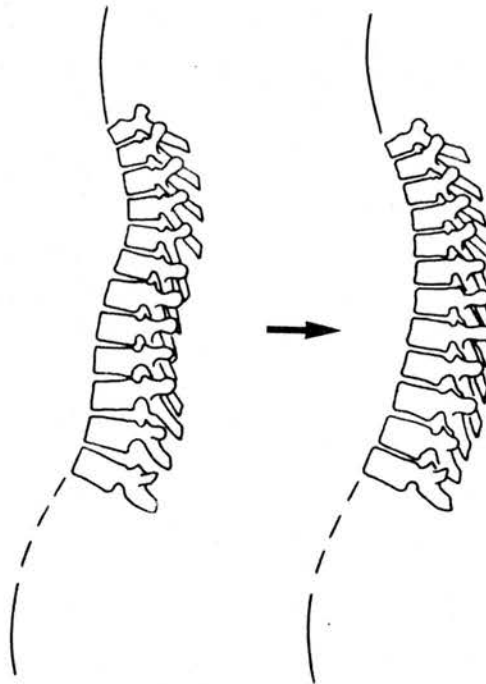


Figure 5.8 Reconstitution of the thoracic kyphosis offers a more physiological approach to treatment.

Table 5.1 Mean Change in AP and Lateral Deformity before and after Surgical Reconstruction of Thoracic Kyphosis in Ten Patients with Adolescent Idiopathic Scoliosis

Type	Before Operation	After Operation
Mean thoracic kyphosis	16.8°	33°
Mean lateral of apex	5° lordosis	12° kyphosis
Mean AP Cobb angle	41.2°	14.8°
Mean RVAD	29.4°	9.6°

of growth to go before peak adolescent growth velocity. The mean deformity magnitudes listed in Table 5.1 refer to our first ten patients. The apical lordosis can be readily converted into a normal kyphosis, and the derotation effect of sublaminar wiring is manifested by a two-thirds correction of the rib-vertebra angle difference. These patients are being closely monitored with regard to spinal shape and subsequent growth.

REFERENCES

1. Nachemson, A. L. Future research in scoliosis. In *Scoliosis* (ed. P. A. Zorab and D. Siegler). Academic Press, London, 1979, pp. 263-270.
2. Brash, J. C. In *Cunningham's Textbook of Anatomy* (ed. J. C. Brash). Oxford University Press, London, 1953, pp. 3-4.
3. Adams, W. *Lectures on the Pathology and Treatment of Lateral and Other Forms of Curvature of the Spine*. Churchill & Sons, London, 1865.
4. Heuer, F. Zur Theorie der Skoliose. *Hessisches Aerzteblatt* No. 18 and ff., 1927. Quoted in P. Wittebol. Idiopathic scoliosis (experimental investigation). *Archivum Chirurgicum Neerlandicum* 8:269, 1956.
5. Somerville, E. W. Rotational lordosis: The development of the single curve. *Journal of Bone and Joint Surgery* 1952, 34B:421-427.
6. Roaf, R. The basic anatomy of scoliosis. *Journal of Bone and Joint Surgery* 1966, 48B:786-792.

7. Deane, G., and Duthie, R. B. A new projectional look at articulated scoliotic spines. Acta Orthopaedica Scandinavica 1973, 44:351-365.
8. Deacon, P., Archer, I. A., and Dickson, R. A. The anatomy and biomechanics of spinal deformity. Journal of Bone and Joint Surgery 1984, 66B:289.
9. Dickson, R. A., and Sevitt, E. A. Growth and idiopathic scoliosis: A longitudinal cohort study. Journal of Bone and Joint Surgery 1982, 64B:385.
10. Dickson, R. A., Lawton, J. O., Archer, I. A., Butt, W. P., Jobbins, B., Berkin, C. R., Bliss, P., and Somerville, E. W. Combined median and coronal plane asymmetry: The essential lesion of progressive idiopathic scoliosis. Journal of Bone and Joint Surgery 1983, 65B:368.
11. Dickson, R. A., Lawton, J. O., Archer, I. A., and Butt, W. P. The pathogenesis of idiopathic scoliosis: Bi-planar spinal asymmetry. Journal of Bone and Joint Surgery 1984, 66B(1):8-15.
12. Lawton, J. O., Butt, W. P., and Dickson, R. A. Experimental idiopathic scoliosis. Journal of Bone and Joint Surgery 1983, 65B:657.
13. Whittle, M. W., and Evans, M. Instrument for measuring the Cobb angle in scoliosis. Lancet 1979, i:414.
14. Roth, M. Idiopathic scoliosis and Scheuermann's disease: Essentially identical manifestations of a neurovertebral growth disproportion. Radiologica Diagnostica (Berlin) 1982, 22:380.
15. Peloux, J. du, Fauchet, R., Faucon, B., and Stagnara, P. Le plan d'elecion pour l'examen radiologique des cyphoscolioses. Revue de Chirurgie Orthopedique 1965, 51:517-524.
16. Brodhurst, B. E. On Lateral Curvature of the Spine. 2nd ed. John Churchill & Sons, London, 1864.
17. Willner, S. Spinal pantograph: A non-invasive technique for describing kyphosis and lordosis in the thoraco-lumbar spine. Acta Orthopaedica Scandinavica 1981, 52:525-529.
18. Dickson, R. A. Scoliosis in the community. British Medical Journal 1983, 286:615-618.
19. Sorenson, K. H. Scheuermann's Juvenile Kyphosis. Munksgaard, Copenhagen, 1964.
20. Moe, J. H., Winter, R. B., Bradford, D. S., and Lonstein, J. E. Scoliosis and Other Spinal Deformities. W. B. Saunders Company, Philadelphia, 1978.
21. Willner, S. A study of growth in girls with adolescent idiopathic structural scoliosis. Clinical Orthopaedics and Related Research 1974, 101:129-135.
22. Low, W. D., Mok, C. K., Leong, J. C. Y., Yau, A. C. M. C., and Liskowski, F. P. The development of Southern

Chinese girls with adolescent idiopathic scoliosis. Spine 1978, 3: 152-156.

23. Leong, J. C., Low, W. D., Mok, C. K., Kung, L. S., and Yau, A. C. M. C. Linear growth in Southern Chinese female patients with adolescent idiopathic scoliosis. Spine 1982, 7:471-475.

24. Delmas, A. Types rachidiens de statique corporelle. Revue de Morphophysiologie Humaine 1951.

25. Stagnara, P., Mauroy, J. C. de, Drau, G., Gonon, G. P., Costanzo, G., Dimnet, J., and Annick, P. Reciprocal angulation of vertebral bodies in a sagittal plane: Approach to references for the evaluation of kyphosis and lordosis. Spine 1982, 7:335-342.

26. Staub, H. A. Eine Skoliotikerfamilie: Ein Beitrag zur Frage der kongenitalen Skoliose und der Heredität der Skoliosen. Zeitschrift für Orthopädie und Chirurgie 1922, 43:1-20.

27. Wynne-Davies, R. Familial scoliosis. In Proc. Symposium on Scoliosis, National Fund for Research into Poliomyelitis and Other Crippling Diseases, London 1965, Action for the Crippled Child Monograph (ed. Zorab, P. A.). Vincent House, London, pp. 36-41.

28. DeGeorge, F. V., and Fisher, R. L. Idiopathic scoliosis: Genetic and environmental aspects. Journal of Medical Genetics 1967, 4:251-257.

29. Cowell, H. R., Hall, J. N., and MacEwen, G. D. Genetic aspects of idiopathic scoliosis. Clinical Orthopaedics and Related Research 1972, 86:121-131.

30. Carr, W. A., Moe, J. H., Winter, R. B., and Lonstein, J. E. Treatment of idiopathic scoliosis in the Milwaukee brace: Long-term results. Journal of Bone and Joint Surgery 1980, 62: 599-612.

31. Blount, W. P., and Moe, J. H. The Milwaukee Brace. Williams & Wilkins, Baltimore, 1973.

32. Luque, E. R. Segmental spinal instrumentation for correction of scoliosis. Clinical Orthopaedics and Related Research 1982, 163:192-198.

SCOLIOSIS : HOW BIG ARE YOU?

Dickson RA.

Orthopaedics; 10: 881-887, 1987.

This paper explained the inaccuracies of measurement of the size of spinal deformities from a postero-anterior projection of the patient and described sagittal spine balance in single and double structural curve patterns.

EDITORIAL

Scoliosis: How Big Are You?

Robert A. Dickson, ChM, FRCS

One of the biggest problems facing surgeons dealing with spinal deformities is assessing the size of the deformities they are seeking to treat. This is also a problem in epidemiological studies of the untreated condition. The optimal situation would be a simple figure expressing the size of the deformity, eg, x° , so that when the patient after treatment had a deformity of one-half x° then a 50% correction would be observed. Unfortunately, this ideal is not achievable at the present time and the traditional methods of measuring curve size do nothing but confuse the issue.

The universal method of measuring curve size is that of Cobb.¹ A postero-anterior (PA) radiograph of the spine is obtained and the upper and lower end vertebrae are identified. The angle, subtended by a line drawn along the upper border of the upper end vertebra and the lower border of the lower end vertebra, is referred to as the Cobb angle of the curve. It is a modification of the method of Ferguson² whose angle was that subtended by lines drawn from the center of the apical vertebra to the center of the upper and lower end vertebra. The use of a special protractor³ makes measurement of the Cobb angle more accurate and avoids the need to cover radiographs with elementary geometrical hieroglyphics. Because a structural scoliosis encompasses rotation, indeed it is defined as such,⁴ the PA view of the patient must necessarily be an oblique view of the deformity, the

degree of obliquity being proportional to the amount of apical rotation (Fig. 1). Thus, the bigger the deformity, the greater the amount of rotation, the more the PA projection of the patient becomes oblique, and the less able it is to describe the true magnitude of the deformity. Thus, a curve which has a Cobb angle of 60° is twice as severe as a curve that measures 30° , while reciprocally, a curve which has been corrected from 60° to 30° has achieved a better correction than 50%. But what is this deformity seen on a PA projection of the patient, and is this measurement of any importance? To answer this important question and to resolve the apparent confusion in the description of spinal deformities, it is necessary to look briefly at the pathogenesis of structural scoliosis.

Perhaps the biggest disservice to scoliosis surgeons has been the discovery of x-rays. A PA radiograph of the patient with a structural scoliosis conveniently reduces to two dimensions a deformity which is clearly happening in three. The lateral spinal curvature now appears dominant and indeed is the definition of scoliosis.⁴ Before the advent of x-rays however, the true nature of the deformity of structural scoliosis had been clearly described as a result of both clinical observation and cadaver dissection.^{5,6} The most important component of the deformity was noted to be lordosis which, in the thoracic region, represented a significant departure from the normal kyphotic configuration. In describing the forward bend test Adams⁶ observed that the lordosis buckled out to the side, enhancing both the rib hump and the scoliosis (Fig. 2); encapsulated in his statement, "Lordosis plus rotation equals lateral flexion." Thus, the lateral curvature, the scoliosis, perceived as being the important component of the deformity on a PA radiograph of a patient, is in reality the secondary deformity. Steindler,⁷ Somer-

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Fig. 1: PA and lateral radiographs of a 4-year-old with an early-onset progressive idiopathic thoracic curve. PA and lateral radiographs of the patient are necessarily oblique views of the deformity. The PA radiograph underestimates the size of the scoliosis while the lateral view shows the spurious appearance of a kyphosis.

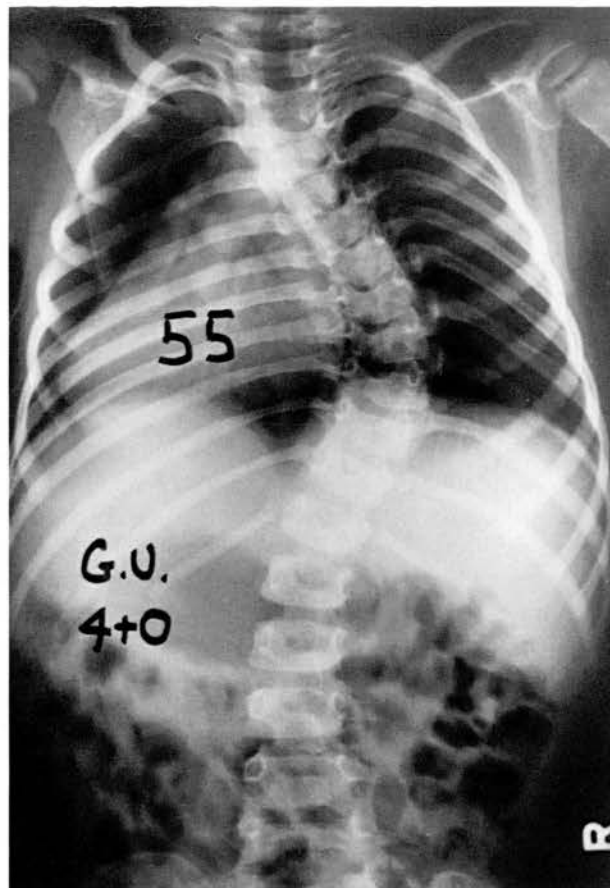


Fig. 1A.

ville,⁸ and Roaf,⁹ the leading orthopedic intellectuals of their generation, readily concurred with the views of Adams and clearly appreciated the true nature of the three-dimensional deformity of structural scoliosis. Unfortunately, their works appear to have been largely ignored.

While working for 10 years at the Nuffield Orthopedic Center in Oxford, I had the privilege of hearing first hand of the work of Somerville, who since retiring from Oxford, has so unselfishly given of his great intellect to my scoliosis research program in Leeds, which I commenced 6 years ago. As a result,

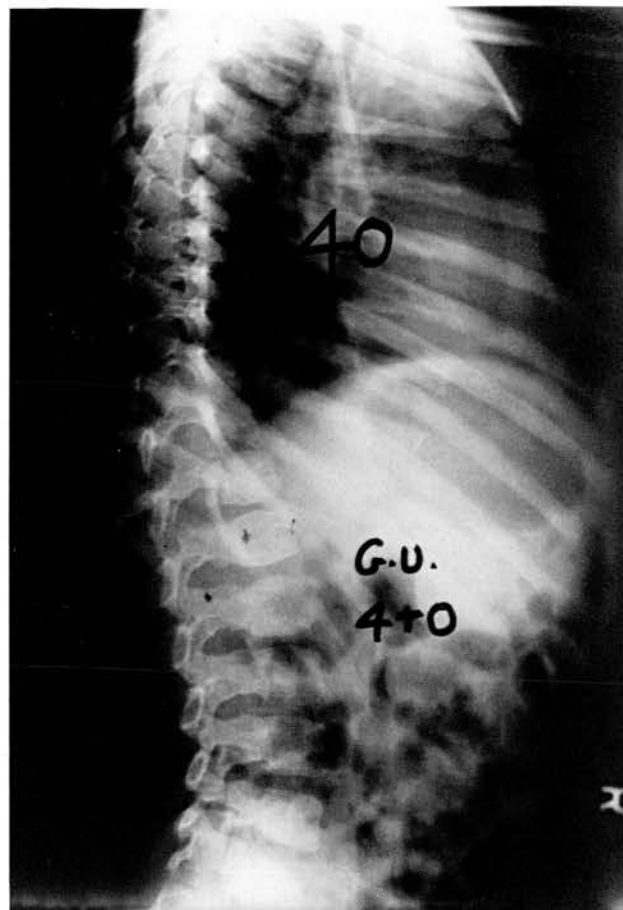


Fig. 1B.

the Leeds Scoliosis Study Group has established, "beyond all reasonable doubt," that lordosis is the essential lesion of every structural scoliosis, no matter where it occurs in the spine and no matter what its etiology (eg, idiopathic, congenital, neuromuscular, neurofibromatous, etc). The following are some of the more important points to emerge.

Our studies have principally concerned structural curves in the thoracic region as the demonstration of lordosis here would imply reversal of normal sagittal shape. We started with the PA radiograph of the patient and far from being confusing, it supplies nearly all the information about the three-dimensional deformity with the exception of its size. As Roaf⁹ clearly pointed out, in every structural curve the posterior elements are rotated into the curve concavity, while the bodies are rotated into the convexity. His elementary geometrical point, that a line drawn down the posterior elements will be shorter

Fig. 2: The forward bending test of Adams.⁶

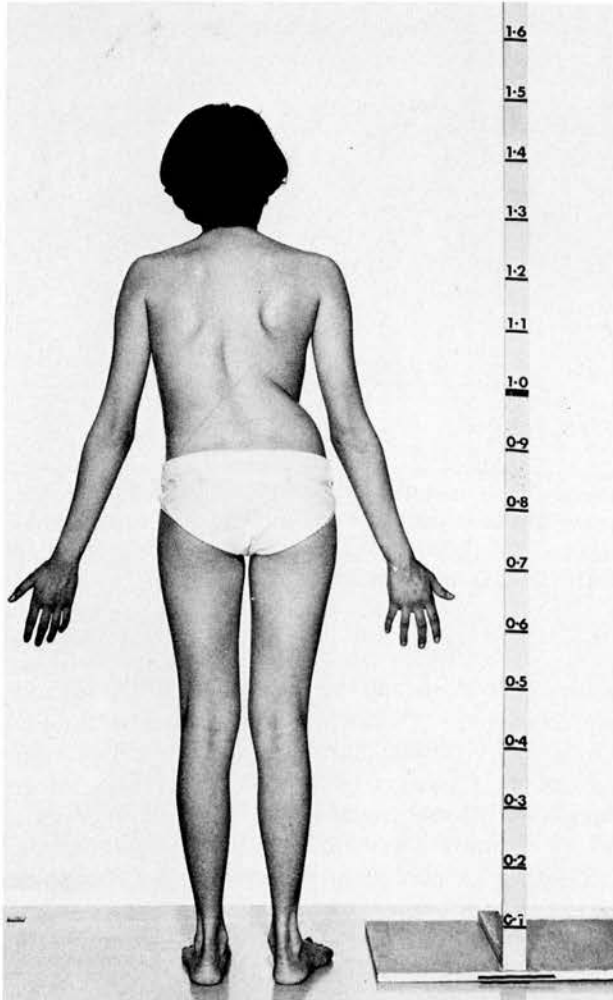


Fig. 2A: The deformity is less obvious when the patient is erect.

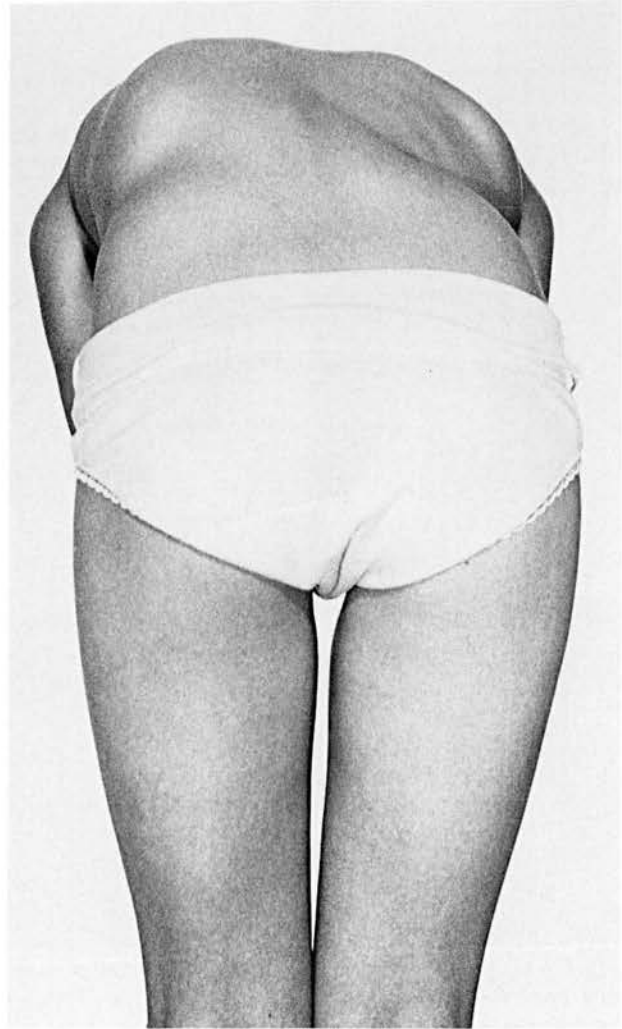


Fig. 2B: On forward bending the fundamental lordosis buckles out to the side to increase the rotational prominence.

than a line drawn down the bodies, and thus the deformity must be lordotic, is indisputable. There is more, however, that can be viewed from the PA radiograph of the patient. Both Cobb¹ and Ferguson,² and many since, have warned that identification of the end vertebrae of a structural curve is solely for measurement purposes and that the first neutral (non-rotated) vertebrae consistently lie one or two above and one or two below the upper and lower end vertebrae respectively. This has been interpreted as meaning that rotation exists in part of the upper and lower compensatory scolioses.⁴ While this statement is important concerning the length of fusion of a

curve, it is dangerously inaccurate with regard to the true nature of the deformity (*vide infra*).

To examine true planar sagittal shape in these spines, we have obtained true lateral projections of each vertebra in the deformity. In our first patients we obtained the amount of rotation by way of computed tomography but later used both the method of Perdriolle¹⁰ and our own digitized version of Nash and Moe¹¹ (both these methods determine the amount of vertebral rotation based on the position of the convex pedicle in relation to the size of the vertebral body on the PA radiograph of the patient). We have thus been able to obtain true PA and lateral projections of the



Fig. 3A: AP radiograph of a 2-year-old with a progressive thoracic curve. There is posterior element rotation into the concavity.



Fig. 3B: True planar (Leeds lateral) projection of the apex of this curve showing the characteristic lordosis.

apical vertebra, or any other part of the spine in which we are interested, by rotating the x-ray beam according to the amount of apical rotation. True PA projections of the curve apex, when the deformity is maximally seen "en face" (referred to as the plan d'élection of Stagnara¹²), now reveal a Cobb angle substantially greater than that obtained from the PA projection of the patient, as the obliquity of the view has been eliminated.¹³ More importantly, when true lateral projections (which we call "Leeds laterals") were obtained, a lordosis extending over the apical four or five segments was constantly observed (Fig. 3) and there was a significant correlation between the

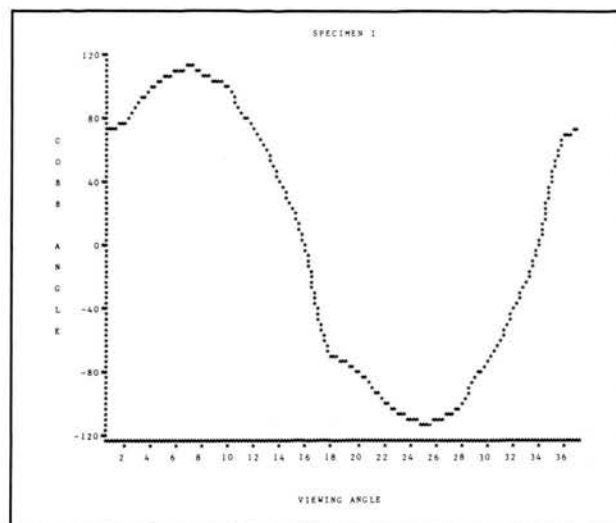


Fig. 4: An articulated spine with an idiopathic thoracic curve has been rotated through 360° and x-rayed at 10° increments through this range. Cobb angle changes with viewing angle in a sine wave form.

degree of lordosis and the Cobb angle of the secondary deformity.¹³⁻¹⁸ Furthermore, when we studied specimens with structural scoliosis obtained from the Royal College of Surgeons of Edinburgh Pathology Museum, and rotated them through 360°, taking radiographs at intervals through this range, the degree of perceived deformity by Cobb angle varied in sine-wave form, the angular appearance depending entirely on the obliquity of the projection (Fig. 4).¹³

The same spurious appearance of deformity size can be confirmed by observing the handle of a brace and bit. When the handle points due east or west, the deformity is seen in maximal relief (the plan d'élection of Stagnara). As it is moved progressively toward either north or south, the size of the deformity diminishes until at the polar positions (the Leeds lateral) no deformity can be perceived. If, however, the handle of the brace and bit in the polar positions is looked at from the side, then the same deformity as the plan d'élection is observed. When the handle is rotated by 45° (north-east, north-west, south-east, south-west) then from front and side a deformity of the same size will be observed. This is precisely the appearance on PA and lateral projections of the patient whose apical rotation measures 45°. These observations clearly cast considerable doubt on the validity of the measurement of Cobb angle from a PA

projection of the patient, but more importantly show the mathematical futility of the way we currently describe our deformity and changes thereof. Changes in Cobb angle with rotation form a geometric progression and not an arithmetic one, yet it is by arithmetic means that we collectively deal with these data. For example, the literature is profuse with reports of, for example, 100 patients with an average Cobb angle of 50° . Deriving averages from non-arithmetic data is not only inadmissible, but means nothing. Similarly, change in Cobb angle as perceived on a PA projection of the patient cannot be expressed arithmetically by percentage. However, the opposite deformity of kyphosis is uniplanar and a lateral projection of the patient yields the true extent of the deformity. Thus, measurement of Cobb angle for kyphosis, along with the taking of means and the calculation of percentages, is quite valid, but not for structural scoliosis.

By taking these true lateral projections of the apex of structural scolioses, and we have now obtained this information on more than 500 patients, we have been able to show that the deformity of idiopathic scoliosis is the opposite of Scheuermann's disease¹⁹ and that the only way of producing a structural curve of the non-paralytic variety in the growing animal is by tethering the spine into a short-segment lordosis.²⁰⁻²² Having observed that the experimentally produced deformity would resolve with growth when the thoracic kyphosis was recreated, we have adopted a more physiological approach to the management of patients with idiopathic thoracic scoliosis in which re-formation of the thoracic kyphosis is the essential part of the instrumentation.¹⁶⁻¹⁸ No other method currently described achieves a greater degree of derotation.²³

There is yet more to learn about the three-dimensional deformity from the PA projection of the patient. While we have confirmed Roaf's simple geometrical point that posterior element rotation into the concavity implies lordosis, the PA projection of the patient also demonstrates that the first few vertebrae in the compensatory scolioses above and below are also rotated in the same direction, ie, toward the concavity of the structural curve. However, and this point is both novel and crucial, these so-called compensatory scolioses are convex to the opposite side of the structural curve (Fig. 5). In these compensatory curves rotation is now such that the posterior elements are directed toward the curve convexity (ie, a rotated kyphosis). Thus, the description of these



Fig. 5: AP radiograph of a patient with a right thoracic idiopathic scoliosis. In the central part of the structural curve the spinous processes point toward the concavity, indicating that this area is lordotic. Above and below this lordotic area (in the region of compensatory scolioses) the spinous processes are still rotated in the same direction but now find themselves on the convexity of these compensatory scolioses. These areas are therefore buckled kyphoses (spinous processes marked with triangles, centers of bodies with dots).

compensatory curves as "compensatory scolioses" is incorrect. As rotated or buckled kyphoses, what they are doing is balancing the three dimensional deformity of rotated lordoscoliosis, which only exists in the apical four or five segments of the structural curve. Indeed, so that the head is centered

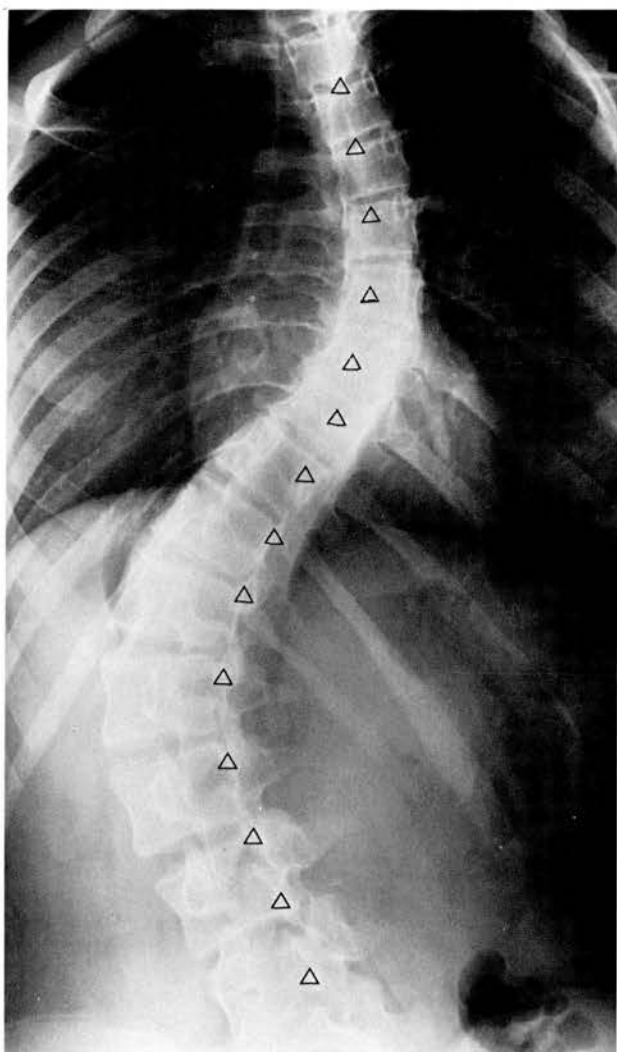


Fig. 6: PA radiograph of a patient with a double structural idiopathic curve. There is no area of intervening kyphosis between the two structural curves, which therefore represent a long area of continuous lordosis with buckled kyphoses above and below (spinous processes marked with triangles).

over the pelvis in the coronal plane (ie, the patient is not leaning forward or backward) the spine must compensate for the structural curve, above and below it. Thus from a PA projection of the patient with a structural thoracic curve the following three-dimensional points can be observed. From above down, the spine is deformed down to the last parallel vertebra (the beginning of the so-called compensatory scoliosis). The next two or three vertebrae are not

rotated and represent an area of pure scoliosis. The next two vertebrae or so represent an area of buckled kyphosis, which then merges into the area of a structural lordoscoliosis but separated by a vertebra which is straight in the sagittal plane. Then below the area of structural lordoscoliosis the sequence is reversed—a vertebra symmetrical in the sagittal plane, an area of buckled kyphosis, an area of pure scoliosis, and a symmetrical spine below.

Double structural curves (eg, right thoracic, left lumbar) are even more interesting on a PA projection of the patient (Fig. 6). There is no intervening area of buckled kyphosis between the two structural lordoscolioses and therefore this double structural deformity pattern indicates an abnormally long area of lordosis. From above, down the sequence for double structural curves is as follows: normal spine, scoliosis, buckled kyphosis, straight segment, structural lordoscoliosis in one direction, structural lordoscoliosis in the other direction, symmetrical vertebra, buckled kyphosis, scoliosis, normal spine.

Thus, for every spinal deformity it is easy to assess the nature of the three-dimensional deformity from the PA projection of the patient, but not its size. In order to demonstrate the true planar profiles of each vertebra from above downward, multiple radiographs would be necessary and, although very low-dose radiographic techniques are available,²⁴ it is both inadvisable and unnecessary to do so. Of course pathological specimens¹³ are invaluable in this respect so that the true nature of the three-dimensional deformity can be appreciated. But how should curve size be measured in the patient? Clearly kyphoses and pure scolioses (eg, non-structural scolioses or those caused by solitary hemivertebrae) present no problem as the spinal column is not buckled and measurement of Cobb angle is entirely satisfactory. There is probably no satisfactory method of assessing the complex series of deformities in the structural scoliotic spine. The best that can be achieved is a compromise. In Leeds we favor the following series of films. A PA radiograph of the patient is taken and the three-dimensional nature of the various deformities noted as above (Fig. 5, 6). We measure the amount of apical vertebral rotation on this film^{10,11} and by adjusting the angle of the x-ray beam obtain a plan d'élection and a Leeds lateral. The former is the best estimate of the maximal deformity of the spinal column and we record this by the Cobb angle. The Leeds lateral is important to us for research purposes and also to judge by how much

sagittal profile locally should be adjusted by instrumentation in order to recreate a normal thoracic kyphosis. Apart from an x-ray of the left hand and wrist for bone age we take no other films and certainly a lateral projection of the patient is a waste of both time and money.

I am honored to have been asked to write this editorial review article, and to solicit articles from Europe. I am indebted to these contributors. Harry Piggott from Birmingham, President of the British Scoliosis Society, is a pioneer of physiological surgery and a superb leader. Michael Edgar from London, a founder member and trustee of the British Scoliosis Society, has one of the biggest scoliosis practices in Europe, is a superb ambassador for British orthopedics and has an enviably unfair share of common sense. John Dove from Stoke-on-Trent, Secretary/Treasurer of the British Scoliosis Society, has brought Mexico to Staffordshire and has sent some of it back in rectangular form. Takis Smyrnis from Athens, leading light of Greek scoliosis, has done much to further our understanding of the epidemiology of this difficult condition. Rene Perdriolle from France seems to have known about the three-dimensional deformity since before the time of Adams. Ian Archer and Philip Deacon from Leeds are but two members of the Leeds Scoliosis Study Group with expert knowledge of the biology and biomechanics of spinal deformities. I am most grateful to them all.

References

1. Cobb JR: Outline for the study of scoliosis. *AAOS Instructional Course Lecture* 1948; 5:261-275.
2. Ferguson AB: The study and treatment of scoliosis. *South Med J* 1930; 23:116-120.
3. Whittle MW, Evans M: Instrument for measuring the Cobb angle in scoliosis. *Lancet* 1979; i:414.
4. Goldstein LA, Waugh TR: Classification and terminology of scoliosis. *Clin Orthop* 1973; 93:10-22.
5. Dick H: A new instrument for the treatment of spinal curve. *Medical Times and Gazette* 1864; II:203-204.
6. Adams W: *Lectures on the Pathology and Treatment of Lateral and Other Forms of Curvature of the Spine*. London, John Churchill and Sons, 1865.
7. Steindler A: *Mechanics of Normal and Pathological Locomotion in Man*. Springfield, IL, Charles C. Thomas, 1935.
8. Somerville EW: Rotational lordosis: The development of the single curve. *J Bone Joint Surg* 1952; 34B:421-427.
9. Roaf R: The basic anatomy of scoliosis. *J Bone Joint Surg* 1966; 48B:786-792.
10. Perdriolle R: *La Scoliose: Son Étude Tridimensionnelle*. Paris, Maloine, 1979.
11. Nash CL, Moe JH: A study of vertebral rotation. *J Bone Joint Surg* 1969; 51A:223-229.
12. du Peloux J, Fauchet R, Faucon B, et al: Le plan d'élection pour l'examen radiologique des cypho-scolioses. *Rev Chir Orthop* 1965; 51:517-524.
13. Deacon P, Flood BM, Dickson RA: Idiopathic scoliosis in three dimensions. A radiographic and morphometric analysis. *J Bone Joint Surg* 1984; 66B:509-512.
14. Dickson RA, Lawton JO, Archer IA, et al: Combined median and coronal plane asymmetry: The essential lesion of progressive idiopathic scoliosis. *J Bone Joint Surg* 1983; 65B:368.
15. Dickson RA, Lawton JO, Archer IA, et al: The pathogenesis of idiopathic scoliosis—Bi-planar spinal asymmetry. *J Bone Joint Surg* 1984; 66B:143-144.
16. Dickson RA, Lawton JO, Archer IA, et al: The pathogenesis of idiopathic scoliosis—Bi-planar spinal asymmetry. *J Bone Joint Surg* 1984; 66B:8-15.
17. Dickson RA, Lawton JO, Butt WP: The pathogenesis of idiopathic scoliosis, in Dickson RA, Bradford DS (eds): *Management of Spinal Deformities*. London, Butterworth, 1984.
18. Dickson RA, Lawton JO, Deacon P, et al: The pathogenesis of idiopathic scoliosis, in Warner JO, Mehta MH (eds): *Scoliosis: Prevention. Proceedings of the Seventh Phillip Zorab Symposium* 1983. New York, Praeger, 1985.
19. Deacon P, Berkin CR, Dickson RA: Combined idiopathic kyphosis and scoliosis: An analysis of the lateral spinal curvatures associated with Scheuermann's disease. *J Bone Joint Surg* 1985; 67B:189-192.
20. Lawton JO, Butt WP, Dickson RA: Experimental idiopathic scoliosis. *J Bone Joint Surg* 1983; 65B:657.
21. Lawton JO, Scott SAJ, Dickson RA: Physiological treatment for idiopathic scoliosis—Experimental basis. *J Bone Joint Surg* 1985; 67B:160.
22. Smith RM, Dickson RA: Progressive experimental scoliosis in the New Zealand white rabbit. *J Bone Joint Surg* 1986; 68B:682.
23. Archer IA, Deacon P, Dickson RA: Idiopathic scoliosis in Leeds—A management philosophy. *J Bone Joint Surg* 1986; 68B:670.
24. Ardran GM, Coates R, Dickson RA, et al: Assessment of scoliosis in children: Low dose radiographic technique. *Br J Radiol* 1980; 53:146-147.

(Calendar continued)

area of use of muscle contraction as an activating force. Concepts of muscle contraction will be taught and will be used in the treatment of the vertebral column and the bony pelvis. The structural diagnostic system will be expanded in dysfunction of the vertebral column and bony pelvis. For further information, contact Sandy Kilbourn, Assistant to the Dean, MSU, East Fee Hall, East Lansing, MI 48824-1316.

September 16-19

The Fourteenth Annual Refresher Course of the International Skeletal Society: Musculoskeletal Disorders will be held in **Cannes, France**. The course is designed to present a review of current knowledge and future trends in musculoskeletal disorders. An interdisciplinary faculty of over 70 radiologists, orthopedic surgeons, and pathologists will consider various aspects of bone and joint disease. Newer concepts and recent developments in the area of skeletal radiology, including magnetic resonance imaging, computed tomography, arthrography, and bone scanning will be provided. For further information, contact Ms. Janice Ford, Convention Manager, International Skeletal Society, 39 Dunlin Way, Erial, NJ 08081, or call (215) 662-6904.

September 18-20

"Tutorial on Level I Myofascial Release Technique" will be held at the Kellogg Center for Continuing Education, Michigan State University, **East Lansing, Michigan**. The course will include intensive exposure to basic concepts of myofascial release manipulative therapy. Emphasis is placed on direct experiences giving participants opportunity to test various forms of motion, and motion changes, and palpate various tissues and forms. For further information, contact Sandy Kilbourn, Michigan State University, East Fee Hall, East Lansing, MI 48824-1316.

September 20-26

London/Paris Third Annual Fall Ultrasound Meeting will be held in **London, England**, September 20-23 and in **Paris, France**, September 23-26. Participants may attend one or both meetings. The meetings are presented by an international faculty. For further information, contact Annual Fall Ultrasound Meeting, Medical Seminars International, Inc, 21915 Roscoe Boulevard, Suite 222, Canoga Park, CA 91304, or call (818) 340-0580, extension 280.

September 26-30

"Tutorial on Level II Manual Medicine Technique (Above Diaphragm)" will be held at the Kellogg Center for Continuing Education, Michigan State University, **East Lansing, Michigan**. This course presents examination, analysis and treatment of the upper extremities, cervicothoracic spine, thoracic cage, throat, and jaw. Postural/structural concepts are expanded into functional and integrative analysis in terms of respiratory, circulatory, neurologic, and fascial models. For further information, contact Sandy Kilbourn, Michigan State University, East Fee Hall, East Lansing, MI 48824-1316.

September 29-30

The Third International Symposium: New Frontier of Biochemistry and Treatment of Stroke and Brain-Spinal Cord Injury will be held in **Firenze, Italy**. Topics will include: Key role of membrane transport ATPase and neurotransmitters intermodulation in NS; ATPase in vitro and in vivo by NMR and EPR, similarity of dismetabolic, cytotoxic and vasogenic. For further information, contact Palazzo dei Congressi, "Sala Verde", telephone: 055-26-22-41.

October 1987

The Third International Symposium: New Frontiers of Bio-

chemistry will be held in **Italy**. This year's topic is Pharmacologic treatment and management of spinal cord injury and paraplegia Satellite meeting on acute craniocerebral trauma and brain ischemia. There is no registration fee. For further information, contact Cincenzo Lombardi, MD, Piazza Garibaldi 48 70021 Acquaviva, Italy.

October 5-9

"Tutorial on Level II Craniosacral Techniques" will be held at the Kellogg Center for Continuing Education, Michigan State University, **East Lansing, Michigan**. The purpose of this tutorial is to review in detail cranial anatomy and specific corrective techniques for membranous strain patterns; to discuss special problems including TMJ, pediatric problems, intrapment syndromes, functional anatomy, and treatment procedures. For further information, contact Sandy Kilbourn, Michigan State University, East Fee Hall, East Lansing, MI 48824-1316.

October 8-10

The International Symposium on Sarcomas will be held at the Innisbrook in **Tarpon Springs, Florida**. For further information, contact James R. Ryan, MD, Department of Orthopedic Surgery, Wayne State University School of Medicine, 4201 St. Antoine 7C, Detroit, MI 48201.

October 10-18

The Twelfth Annual International Body Imaging Conference will be held in **Maui, Hawaii**. The internationally renowned faculty will present a correlative approach to computed tomography, ultrasonography, digital radiography, conventional radiology, and magnetic resonance imaging. For further information, contact the Annual Body Imaging Conference, Department of Radiology, Nu Med

(continued on page 896)

THE ANATOMY OF SPINAL DEFORMITY : A BIOMECHANICAL ANALYSIS.

Deacon P, Archer IA, Dickson RA.

Orthopaedics; 10: 897-903, 1987.

In addition to analysing the anatomy and biomechanics of normal spinal shape it was explained why deformities occurred on an anatomical and biomechanical basis.

The Anatomy of Spinal Deformity: A Biomechanical Analysis



Philip Deacon, ChB, FRCS*

Ian A. Archer, ChB, FRCS†

Robert A. Dickson, ChM, FRCS§

ABSTRACT: The essential lesion in idiopathic scoliosis is a lordosis at the curve apex. For the rotational instability provided by a lordosis to progress, asymmetry must be present in another plane. A biomechanical analysis of spinal shape demonstrates a delicate balance between the median, transverse, and coronal planes. The normal cervical and lumbar lordoses which are inherently rotationally unstable are protected by: a) prismatic shaped vertebral bodies with their bases anterior, b) considerable available intersegmental flexion, and c) powerful posterior soft tissue support. In contrast, the thoracic vertebral bodies are shaped as prisms with their apices anterior. This rotationally unstable configuration is protected by a kyphosis with the axis of spinal rotation situated anteriorly. The thoracic vertebral prisms are asymmetric, their apices lying to the right of the median plane. In the presence of a lordosis the apices of the prisms will be directed toward the right producing a right-sided scoliosis. Any significant degree of left-sided coronal asymmetry can override the influence of the transverse plane and therefore left-sided curves are not uncommon. Thoracic idiopathic scoliosis is located at T8/T9 and the lordosis here is often an upward

continuation of the normal lumbar lordosis such that the asymmetric thoracic prisms are no longer protected.

Introduction

There are two basic types of progressive spinal deformity: lordoscoliosis and kyphosis. Both share the common feature that the essential lesion is an alteration of spinal shape in the median (sagittal) plane. In a lordoscoliosis the underlying lesion is a lordosis which moves out to the side producing the secondary deformity, scoliosis, in the coronal plane.¹⁻⁶ Rotation always occurs in the same direction, the vertebral bodies rotating to the convexity of the scoliosis and the posterior elements to the concavity confirming that it is a lordosis which has rotated (Fig. 1). The established deformity is therefore triplanar. A kyphosis however, exists only in one plane. The anteriorly placed axis of spinal rotation protects the kyphosis from rotation which does not therefore occur. The normal spine is not symmetrical in any of its three planes, median, coronal, or transverse and thus possesses inherent potential to develop deformity which is expressed when normal protective mechanisms become deficient. This anatomical and biomechanical analysis of spinal shape demonstrates how the normal spine is protected from deformity and explains the pathogenesis of idiopathic curves and their predilection for certain sites and direction.

Anatomical and Biomechanical Analysis

Thoracic Region

Anatomy. By convention the thoracic spine extends from T1 to T12 but this is not so anatomically. Typical "thoracic" vertebrae extend only from

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Fig. 1: Anteroposterior radiograph of idiopathic scoliosis showing lateral curvature and vertebral rotation. The spinous processes are directed toward the concavity demonstrating that it is a lordosis which has rotated.

T3 to T9 while the first two and last three thoracic vertebrae resemble typical cervical and lumbar vertebrae respectively, and belong to their lordoses.^{7,8} Anatomically the thoracic spine extends from T3 to T9 and is smoothly kyphotic, a curvature which is produced by vertebral shape in the median plane. The vertebral bodies have reduced height anteriorly relative to posteriorly while the thin intervertebral discs are symmetrical. In the transverse plane a typical thoracic vertebra is heart-shaped having a greater anteroposterior depth than lateral width (a prism with its apex anteriorly). The prism however, is asymmetric in the transverse plane being flattened on the left side from T4 to T9 by the close apposition of the

descending thoracic aorta (Fig. 2), this effect being most marked when the thoracic kyphosis is relatively reduced. Pedicle asymmetry noted in the second half of the first decade of life has also been attributed to the influence of the aorta.⁹ The first two and last three thoracic vertebrae lose their kyphotic and prismatic shape as they become closer to their cervical and lumbar neighbors. The intervertebral facet joints lie on the arc of a circle, the center of which lies anteriorly in the vertebral body, and therefore permit a large range of rotation in this region.

Intervertebral disc height is, however, limited in the thoracic spine and the limits of forward flexion are soon reached with additional motion restriction from the attached thoracic cage. Gross anatomists frequently described a curvature of the thoracic spine to the right in the coronal plane which they attributed to aortic pressure or upper limb dominance.⁷⁻¹² Epidemiological surveys of spinal shape, however, indicate that small degrees of coronal plane curvature are randomly directed, tending to become right sided only when substantial.¹³⁻¹⁶

Biomechanics. When a prismatic structure is flexed toward its base it is rotationally stable, but when the apex is under compression it is extremely vulnerable to rotation (Fig. 3). Accordingly the thoracic region is normally protected by the presence of a kyphosis which places the axis of spinal rotation anterior to the vertebral bodies which, therefore, do not normally rotate on flexion. Further protection from rotation is provided by the attached thoracic cage. Beyond the limits of normal flexion of the thoracic spine buckling occurs and the asymmetry of the prismatic shape in the transverse plane directs this to the right (Fig. 4). Lateral flexion in this situation is associated with rotation such that the spinous processes move to the convexity of the curve, the axis of rotation, and the center for movement at the intervertebral joints both lying anteriorly.

Biomechanics of Deformity. Once the vulnerably shaped asymmetric thoracic prisms are no longer protected by a kyphosis they become rotationally unstable (Fig. 4). The axis of spinal rotation in flexion now lies posterior to the apex of the prism which must move to the side. Direction would be random unless governed by pre-existing asymmetry in the transverse or coronal planes and there is evidence of both. Constant transverse plane asymmetry caused by the descending thoracic aorta directs curves to the right and protects minor degrees of left sided coronal plane curvature from progressing. In addition, the

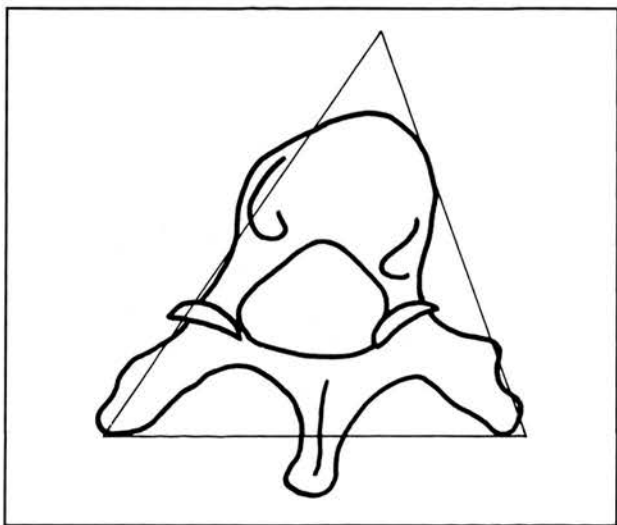


Fig. 2: A typical thoracic vertebra is shaped like a prism with its apex directed anteriorly. The apex of the prism lies to the right of the midline due to the pressure effect of the descending thoracic aorta.

axis of rotation of the lordotic apex lies posteriorly and this limits intervertebral rotation in this region, this movement occurring between vertebrae lying above and below the apex of the deformity. Conversely when the lesion is one of increased kyphosis then the deformity does not include any significant rotation, though the compensatory lordoses above and below the kyphotic region are the sources of the scolioses associated with Scheuermann's disease.¹⁷

Cervical and Lumbar Regions

Anatomy. While the cervical region extends by convention from C1 to C7 and the lumbar region from L1 to L5, the characteristics of typical cervical and lumbar vertebrae are also found at the beginning and end of the thoracic region.^{7,8} In both cervical and lumbar regions lordoses are normal and are due to a combination of both disc and vertebral shape in the median plane. Intervertebral disc spaces are wide and the available flexion is considerable. The fifth lumbar vertebra is markedly lordotic, and to a lesser extent the fourth and third are also. The second is symmetrical and the first is slightly kyphotic. In the transverse plane the vertebrae are broader from side to side than front to back in a ratio of 3:2 in the cervical region and greater still in the lumbar region. The descending thoracic aorta becomes the abdominal aorta in the midline over the body of T12 and

Fig. 3: Radiographs of a prismatic spinal model during flexion. Right [C] is an anteroposterior projection, Left [L] is a lateral projection.

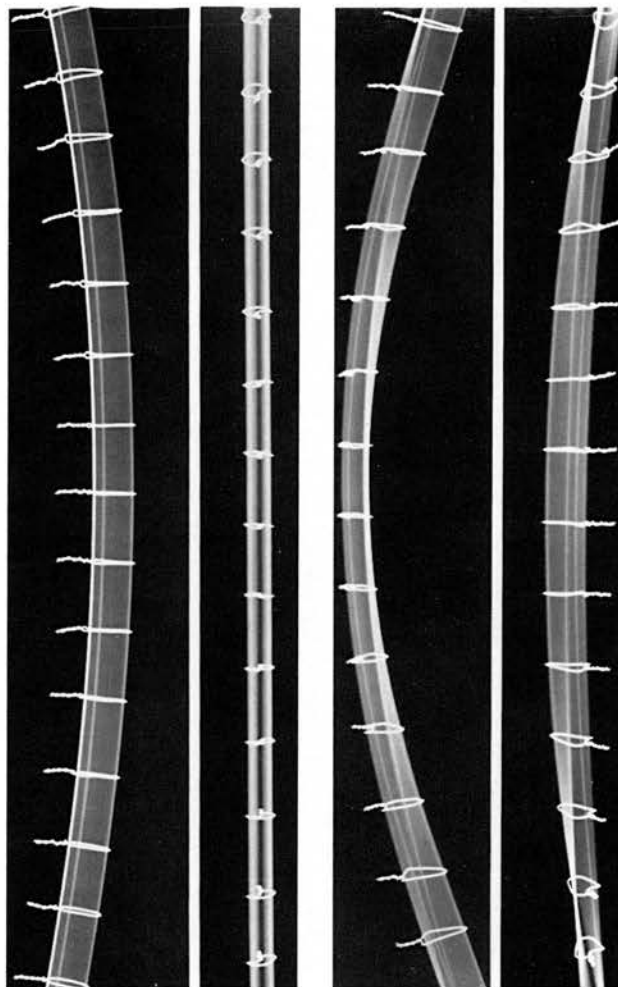


Fig. 3A: Flexion toward base, note absence of rotation.

Fig. 3B: Flexion toward apex, note marked degree of rotation.

terminates at its bifurcation to the left of the median plane over the body of L4. There is little evidence of asymmetry of vertebral shape in the transverse plane in the lumbar region.⁹

The integrity of both cervical and lumbar lordoses is maintained by strong posterior muscular, ligamentous, and fascial systems which are more powerful in these regions than anywhere else in the axial skeleton. In the lumbar spine the orientation of the intervertebral facet joints is such as to prevent rotation between individual vertebrae. The upper facets of L4

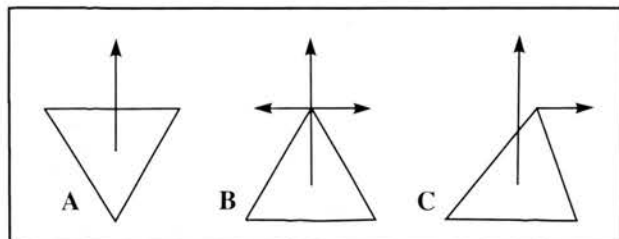


Fig. 4: Rotational behavior of prismatic structures during flexion. A: Flexion toward the base, a stable configuration. B: Flexion toward the apex, rotational instability generated. C: Asymmetric prism flexion toward the apex, rotational instability directed to one side only.

and L5 however face posteriorly as well as medially and therefore permit some rotation about an axis lying behind the vertebral bodies.

Biomechanics. The cervical and lumbar lordoses are inherently unstable to rotation during flexion as the axis of rotation lies posterior to the vertebral bodies. Protection against rotation is considerable. The vertebral bodies in the transverse plane are shaped as prisms with their bases anteriorly (Fig. 5) and the increased height of the intervertebral discs allows considerable flexion to occur before the limit of forward motion is reached. This is controlled by the symmetrical payout of powerful posterior soft tissue structures. There is no obvious asymmetry of vertebral body shape in the transverse plane to direct rotational instability.

Biomechanics of Deformity. In the cervical and lumbar regions lordoses already exist providing substantial potential for the development of a rotational deformity should the normal protective mechanisms become deficient, though asymmetry in the transverse or coronal planes is required to direct rotation. While there is little evidence of static transverse plane asymmetry, the presence of the abdominal aorta, a relatively rigid structure due to the high pressure of the arterial blood contained within it, to the left of the median plane in front of the base of the lumbar prism confers a dynamic form of transverse plane asymmetry. This presence causes the prism to rotate to the left, rotation to the right being prevented. Epidemiological surveys have shown that many apparently normal children have a coronal plane curvature in the lumbar region with a significant left sided preponderance for curves of all magnitude.¹³⁻¹⁶ Any asymmetrical failure of the soft tissue

Fig. 5: Cervical and lumbar vertebrae are shaped as prisms with their bases directed anterior.

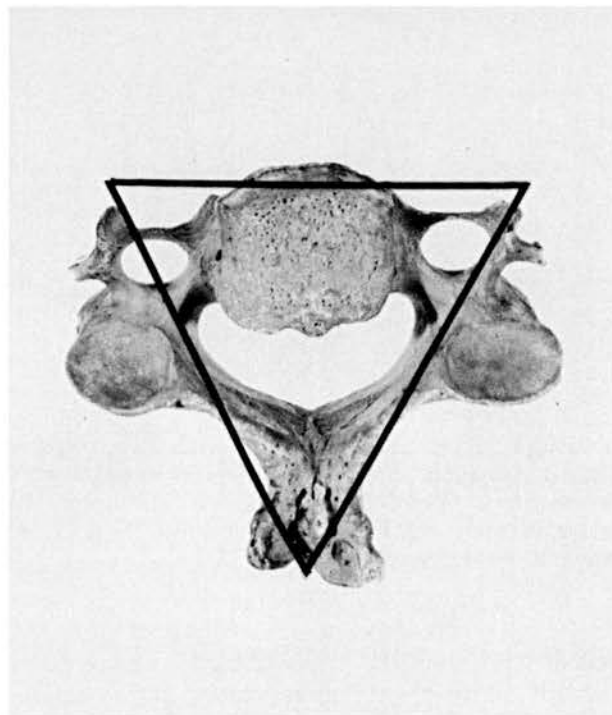


Fig. 5A.

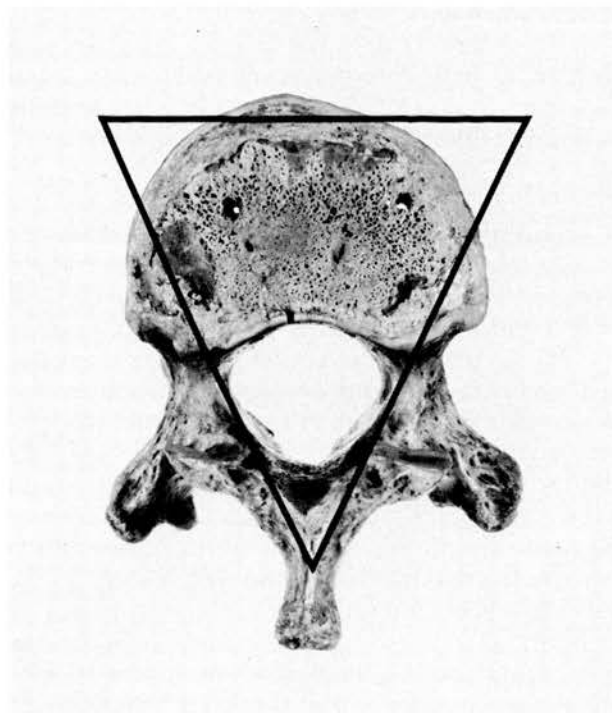


Fig. 5B.

supportive mechanism provides immediate rotational instability compounded by the adverse effect of gravity (Fig. 6). When deformity does occur, the restriction of intervertebral rotation produced by the orientation of the facet joints causes the lumbar curve as a whole to rotate to the side. Compensatory derotation occurs above at the thoracolumbar junction and below at the L4/L5 level where the direction of the facet joint surfaces permits some rotation.

Discussion

There is considerable evidence of potential rotational instability in the normal spine which is not strictly symmetrical in any of the three planes. Where rotational potential exists however, other anatomical factors normally protect accordingly. Normal spinal shape is therefore very delicately balanced and it takes little to create a progressive deformity. The shape of the spine in the median plane provides the key to the development of progressive spinal deformities. Idiopathic scoliosis is the most common progressive spinal deformity and the scoliotic component is entirely secondary. Adams in 1865 stated that lordosis plus rotation equals lateral flexion and this was restated subsequently by Heuer, Somerville, and Roaf.¹⁻⁴ This must be the case as the axis of spinal rotation of a lordosis is posterior to the vertebral bodies which rotate away from the median plane in such a direction that the posterior elements are directed toward the concavity, a constant finding in idiopathic scoliosis. When a kyphosis buckles the posterior elements are directed toward the convexity. True lateral projections of the curve apex in idiopathic scoliosis always demonstrate a lordosis or lordotic tendency.^{5,6} This is not necessarily an area of lordosis between upper and lower areas of kyphosis but is often an upward continuation of the lumbar lordosis such that the area T7, T8, T9 is lordotic when it should be kyphotic. Such a radical alteration in median plane shape is not necessary in the cervical or lumbar regions which already have inbuilt lordoses. A lordosis however would still theoretically be in equilibrium and no rotation would occur if it were not for asymmetry in one or both of the other two planes.

In the thoracic region static transverse plane asymmetry caused by the pressure of the descending thoracic aorta provides the necessary bi-planar instability and directs rotation. The apex of the asymmetric prism rotates to the right producing a right sided scoliosis and a right rib hump with the pos-

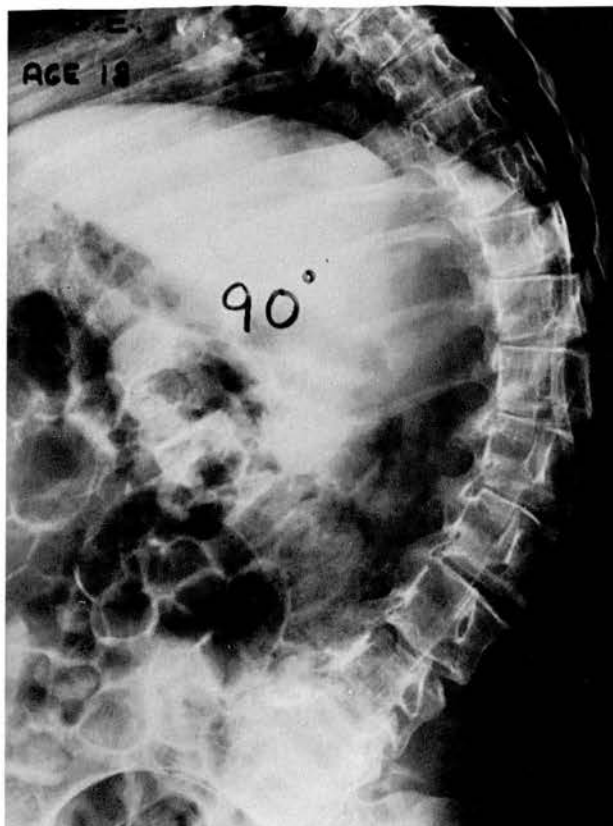


Fig. 6: Typical collapsing lordoscoliosis seen when neuromuscular disease produces asymmetric failure of the soft tissue restraints to the rotational instability of the lordotic lumbar spine.

terior elements directed toward the concavity. Because flexion provides the important deforming force the rotation increases with forward flexion as the apex of the prism is forced to move further from the median plane and conversely decreases during extension. Thoracic idiopathic scoliosis is modal in regard to site at the T8 area. Since the normal thoracic kyphosis ends at the T9/T10 junction only a minimal extension upward of the lumbar lordosis is necessary to produce rotational instability in this region, the vertebral prisms with their apices anterior no longer being protected by a kyphosis. This flattening of the lateral profile of patients with idiopathic scoliosis is a constant clinical feature.^{5,18} This produces an uncoiling effect which explains why children with idiopathic scoliosis are marginally taller than their straight backed peers but do not grow faster and demonstrate no other growth abnormalities.¹⁹

More thoracic curves are convex to the left than would be anticipated from the prevalence of dextrocardia or situs inversus and this is because true coronal plane asymmetry is also an important factor as screening programs demonstrate.¹³⁻¹⁶ There are as many left sided curves of small magnitude as there are right sided and if the degree of coronal plane curvature is sufficient to override the influence of the transverse plane then the coronal plane asymmetry will govern the direction of curvature.

Between the thoracic area where lordoscolioses tend to be convex to the right and the lumbar region where they tend to be convex to the left, there are an equal number of left and right sided curves. This is because in the thoracolumbar region the descending thoracic aorta lies in the median plane and favors no particular rotational direction. Lower down the abdominal aorta lies to the left of the median plane and while not producing sufficient pulsatile pressure to deform these vertebrae in the transverse plane, it provides an important dynamic form of transverse plane asymmetry. At the limits of forward flexion of the lumbar lordosis the presence of the abdominal aorta on the left side of the base of the prism prevents rotation to the right and directs it to the left side. In addition, in the lumbar region, coronal plane curvatures are important. Epidemiological surveys have demonstrated that coronal plane curvatures in the lumbar region are predominantly left sided at all magnitudes and therefore both the coronal and transverse planes favor a left sided direction. When the important soft tissue supportive system maintaining the integrity of the lumbar lordosis fails asymmetrically, as it commonly does in neuromuscular conditions, then a collapsing lordoscoliosis is produced, compounded by the adverse influence of gravity. Indeed this is the typical curve pattern of such unfortunate individuals. Screening programs have also harvested children with nonstructural lumbar curves randomly directed as a result of a minor degree of leg length inequality.¹⁶ These have been detected by the presence of a mild loin hump on the forward bending test which is due to the leg length inequality producing the mild degree of coronal plane spinal curvature necessary to produce rotation in a pre-existing lordosis. In the cervical region idiopathic scoliosis does not occur and what have been termed cervical curves are in fact apical at T3. This is another important junctional area where the cervical lordosis runs into the thoracic kyphosis. If the lordosis involves the third thoracic vertebra then

its prismatic shape, apical anteriorly, provides the necessary rotational instability.

Idiopathic kyphosis (Scheuermann's disease) is the opposite lesion to idiopathic scoliosis in the median plane with an increase in the normal kyphosis due to the vertebral bodies in the median plane being wedge shaped, with reduced height anteriorly, and relatively increased height posteriorly.^{5,20} The axis of spinal rotation in flexion is anterior to the vertebral bodies which are protected from rotation. When the limits of forward flexion are reached however buckling can occur and this is directed to the right because of the transverse plane asymmetry. A very mild rib hump may be visible on the right side at the curve apex but an x-ray determines that the spinous processes are rotated toward the convexity. More importantly in 60% of all cases of idiopathic kyphosis there is a true lordoscoliosis above or below the kyphotic region.¹⁷ This is due to the compensatory lordosis which occurs above or below an area of increased kyphosis and in itself provides rotational instability.

Significant rotational progression does not occur because the nearby kyphosis directs the axis of spinal rotation in flexion more anteriorly and thereby protects it. Indeed the apex of such a compensatory idiopathic scoliosis is always a few vertebrae away from the apex of the kyphosis confirming that it is not the kyphotic area that has rotated. In addition, the older age of onset of Scheuermann's disease reduces the effect of progression with growth. Furthermore, such observations of idiopathic scoliosis and kyphosis occurring in the spine of the same, otherwise normal, child separated only by three or four vertebrae lends strong support to the concept that the underlying pathological process is the same in each. When children with a normal thoracic kyphosis and no scoliosis flex fully, a slight degree of buckling is often observed giving rise to a very mild rib hump, but when the child assumes the erect position no lateral curvature is discernible clinically or radiographically.²¹ This is the effect of the normally asymmetric thoracic prisms buckling slightly and is what is being detected by ultra-sensitive screening tests. Caution must therefore be applied to forward bending tests carried out for screening purposes.

References

1. Adams W: *Lectures on the Pathology and Treatment of Lateral and Other Forms of Curvature of the Spine*. London, J. Churchill and Sons, 1865.

2. Heuer F: Zur Theorie der Skoliose. Hessisches Arzteblatt 1927, No. 18 (cited by Wittebol P: Idiopathic scoliosis. *Arch Chirg Neerland* 1956; 8:269-279).
3. Somerville EW: Rotational lordosis the development of the single curve. *J Bone Joint Surg* 1952; 34B:421-427.
4. Roaf R: The basic anatomy of scoliosis. *J Bone Joint Surg* 1966; 48B:786-792.
5. Dickson RA, Lawton JO, Archer IA, et al: The pathogenesis of idiopathic scoliosis: Biplanar spinal asymmetry. *J Bone Joint Surg* 1984; 66B:8-15.
6. Deacon P, Flood BM, Dickson RA: Idiopathic scoliosis in three dimensions: A radiographic and morphometric analysis. *J Bone Joint Surg* 1984, in press.
7. Frazer JES, Breathnach AS (ed): *The Anatomy of the Human Skeleton*, ed 4. London, Churchill Ltd, 1940.
8. Inkster RG: Osteology, in Brash JC (ed): *Cunningham's Text-Book of Anatomy*, ed 9. Oxford University Press, 1953.
9. Taylor JR: Scoliosis and growth. Patterns of asymmetry in normal vertebral growth. *Acta Orthop Scand* 1982; 54:96-602.
10. Sabatier: *Traite Complete d'Anatomie*. Paris, 1777.
11. Cruveilhier: *Descriptive Anatomy*. Madden W (trans). London, Whittaker, 1841.
12. Farkas A: Physiological scoliosis. *J Bone Joint Surg* 1941; 23:607-627.
13. Brooks HL, Azen SP, Gebert E, et al: Scoliosis: A prospective epidemiological study. *J Bone Joint Surg* 1975; 57A:968-972.
14. Lonstein JE: Screening for spinal deformities in Minnesota schools. *Clin Orthop* 1977; 126:33-42.
15. Rogala EJ, Drummond DS, Gurr J: Scoliosis: Incidence and natural history. *J Bone Joint Surg* 1978; 60A:173-176.
16. Dickson RA: Scoliosis in the community. *Br Med J* 1983; 286:615-618.
17. Deacon P, Berkin CR, Dickson RA: Idiopathic combined kyphosis and scoliosis: Scheuermann's disease revisited. *J Bone Joint Surg* 1984, in press.
18. Willner S: Spinal pantograph: A noninvasive technique for describing kyphosis and lordosis in the thoraco-lumbar spine. *Acta Orthop Scand* 1981; 52:525-529.
19. Dickson RA, Sevitt E: Growth and idiopathic scoliosis: A longitudinal cohort study. *J Bone Joint Surg* 1982; 64B:385.
20. Roth M: Idiopathic scoliosis and Scheuermann's disease: Essentially identical manifestations of a neuro-vertebral growth disproportion. *Radiol Diagn* 1982; 22:380-391.
21. Burwell RG, James NJ, Johnson F, et al: Standardized trunk asymmetry scores: A study of back contour in healthy schoolchildren. *J Bone Joint Surg* 1983; 65B:52-463.

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VERTEBRAL SHAPE IN THE MEDIAN SAGITTAL PLANE IN IDIOPATHIC THORACIC
SCOLIOSIS. A STUDY OF TRUE LATERAL RADIOGRAPHS IN 150 PATIENTS.

Deacon P, Dickson RA.

Orthopaedics; 10: 893-895, 1987.

By taking spinal radiographs 90° to the amount of apical rotation a true lateral projection of the apex of thoracic curves was obtained in 150 patients. Vertebral body shape was also measured and shown to be significantly longer at the front than the back in the apical region demonstrating that the lordosis existed as a result of an abnormality in vertebral shape rather than the intervening discs.

Vertebral Shape in the Median Sagittal Plane in Idiopathic Thoracic Scoliosis

A Study of True Lateral Radiographs in 150 Patients



Philip Deacon, ChB, FRCS*
Robert A. Dickson, ChM, FRCS†

Introduction

Idiopathic scoliosis is defined as a lateral curvature of the spine with vertebral rotation in the absence of any congenital spinal anomaly or musculoskeletal condition. It is the presence of rotation, which is important in producing the deformity, that confuses our understanding of both the condition and its pathogenesis. The postero-anterior (PA) radiograph of a patient with idiopathic scoliosis (Fig. 1) reveals that it is in fact only a PA projection of the undeformed spine above and below the region of the scoliosis, which, as a consequence of the presence of rotation, is seen obliquely.

Much about the true nature of the deformity can be appreciated, however, and there are two main points. Vertebral rotation is in a constant direction no matter where in the spine the idiopathic scoliosis occurs, with vertebral body rotation to the curve convexity and posterior element rotation to the curve concavity. In the thoracic region this must imply the presence of a lordosis throughout the curve, since a rotated kyphosis would have the posterior elements on the curve convexity. Furthermore, as pointed out by Roaf,¹ it is elementary geometry, if rotation is such that the spinous processes remain closer to the midline than the bodies, then the front of the spine is longer than the back, ie, a lordosis must be present. Just as the PA projection of the patient is an oblique view of the deformity according to the amount of vertebral rotation, so a lateral view of the patient will

also be an oblique view of the same deformity. If the scoliosis is of sufficient magnitude the spurious appearance of a kyphosis is produced.²

Clinical observations³ of a lordosis at the curve apex in patients with idiopathic scoliotic deformities were first made over a century ago, well before the advent of radiology, and studies using spinal pantography.⁴ These studies show that children with idiopathic thoracic scoliosis have an overall thoracic kyphosis which is significantly less than normal. These observations and their significance have until recently largely been ignored. A preliminary study of the lateral spinal profiles of 70 patients with idiopathic thoracic scoliosis attending the scoliosis clinic at Leeds confirmed this flattening of the thoracic kyphosis.⁵ However, these latter measurements were made on standard lateral radiographs of the patient and not on true lateral radiographs of the rotated deformity. Accordingly, therefore, we set out to investigate spinal shape in the median sagittal plane as seen in true lateral radiographs of the curve apex in patients with idiopathic thoracic scoliosis.

Materials and Methods

One hundred fifty consecutive patients with idiopathic thoracic scoliosis referred to the Leeds Scoliosis Clinic were studied. PA and lateral radiographs were obtained for each patient using a standard radiographic technique. On each PA radiograph the amount of rotation of the apical vertebra in the curve was measured by the method of Perdriolle.⁶ Using this information the patient was turned through a corresponding arc and a true derotated lateral of the apex of the scoliosis was taken. The series was completed by a radiograph of the left hand and wrist taken for estimation of bone age by the method of Tanner et al.⁷

From these radiographs the size of the scoliotic deformity and the sagittal plane curvatures were

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Fig. 1: PA radiograph of an idiopathic thoracic scoliosis showing a lateral curvature with posterior element rotation to the concavity (a lordotic configuration).

measured using Whittle's⁸ adaptation of Cobb's method. On the true lateral radiographs the anterior and posterior heights of the apical three vertebral bodies and the intervening discs were measured using Vernier calipers. The effect of differences in magnification resulting from variations in patient size and radiographic technique was eliminated by the calculation of an index of wedging (anterior height/posterior height). Statistical significance was determined using Student's t-test for matched pairs and by calculation of the correlation coefficients.

Results

There were 43 males and 107 females. Their mean age was $13.4 \text{ years} \pm 3.2 \text{ years}$ (2 SD), range 7.2 to 19.5 years. The mean Cobb angle of their scolioses was $45^\circ \pm 25^\circ$, range 10° to 140° , and the mean apical rotation was $21^\circ \pm 11^\circ$, range 2° to 90° . Measurements of the lateral radiographs of the patient showed the mean overall thoracic kyphosis to be $20^\circ \pm 11^\circ$, and true lateral radiographs of the apices of the deformities demonstrated that in all cases the spine was lordotic with a mean of $7^\circ \pm 4.5^\circ$, range 1° to



Fig. 2: True lateral radiograph of the curve apex showing lordotic wedging with posterior Schmorl node formation.

31° , over the apical three vertebral levels (Fig. 2).

For all three vertebrae at the apex of the deformity, anterior body height was significantly greater than posterior body height ($P < .001$), and the indices of wedging were greater than one. In many cases this reversed wedging of the normally kyphotic thoracic vertebrae was associated with marked posterior Schmorl's node formation and vertebral end plate changes (Fig. 1B). Measurements of intervertebral disc height revealed that these were either symmetrical or with posterior disc height minimally greater than anterior disc height.

There was a significantly positive correlation between the mean index of vertebral body wedging and the size of the apical lordosis ($P < .001$), though the degree of wedging was not related to either chronological or skeletal age ($P > .05$). The size of the apical lordosis was significantly positively correlated with both the degree of apical rotation ($P < .001$) and the Cobb angle ($P < .001$), though was significantly negatively correlated with the size of the overall thoracic kyphosis ($P < .05$).

Discussion

True lateral projections of the curve apex demonstrated the presence of a lordosis in all cases of idiopathic thoracic scoliosis. The lordosis was present at bony level and resulted from reversed wedging of the normally kyphotic thoracic vertebrae. The significantly positive correlations between the size of the lordosis and both the size of the scoliosis and the amount of apical rotation indicate the fundamentally important role of the lordosis in the production of the deformity. The larger the lordosis the more it had rotated and the greater the lateral curvature produced.

A biomechanical analysis of the effect of a short lordotic segment in a normally kyphotic region of the spine demonstrates⁵ that forward flexion produces a tightening of the posterior structures and a reactive force, the horizontal component of which, acting at a distance from the mid-line, will produce a spinning moment such that the posterior elements rotate to the curve concavity and the vertebral bodies to the curve convexity. This is exactly the situation seen in patients with idiopathic scoliosis and that which results from the creation of a short-segment lordosis in an experimental animal model.⁹

The finding that the apical lordosis was due to an alteration in spinal shape at bony level, with the frequent observation of posterior Schmorl's node formation and vertebral end plate changes similar to those seen in Scheuermann's disease, suggests a

common etiology for the kyphotic condition and idiopathic thoracic scoliosis. Thus vertebral end plate irregularity and Schmorl's node formation occurring anteriorly in the thoracic vertebrae toward the end of the adolescent growth period when the thoracic kyphosis is increasing results in Scheuermann's disease (idiopathic kyphosis).

Similar end plate changes situated posteriorly, and occurring in the pre-adolescent period when the thoracic kyphosis is at a minimum, lead to the creation of a short-segment lordosis with the potential to progressively rotate with repeated flexion cycles and continued growth to produce the typical idiopathic scoliotic deformity. This study confirms that in every case of idiopathic thoracic scoliosis there is a lordosis at the curve apex and that this is due to a reversed wedging of the vertebral bodies in the sagittal plane.

References

1. Roaf R: The basic anatomy of scoliosis. *J Bone Joint Surg* 1966; 48B:786-792.
2. Deacon P, Flood BM, Dickson RA: Idiopathic scoliosis in three dimensions. A radiographic and morphometric analysis. *J Bone Joint Surg* 1984; 66B:509-512.
3. Adams W: *Lectures on the Pathology and Treatment of Lateral and Other Forms of Curvature of the Spine*. London, Churchill & Sons, 1865.
4. Willner S, Johnsson B: Thoracic kyphosis and lumbar lordosis during the growth period in boys and girls. *Acta Paediatr Scand* 1983; 72:873-878.
5. Dickson RA, Lawton JO, Archer IA, et al: The pathogenesis of idiopathic scoliosis—Bi-planar spinal asymmetry. *J Bone Joint Surg* 1984; 66B:8-15.
6. Perdriolle R: *La Scoliose: Son Étude Tridimensionnelle*. Paris, Maloine, 1979.
7. Tanner JM, Whitehouse RH, Takaishi M: Standards from birth to maturity for height, weight, height velocity and weight velocity: British children 1965 II. *Arch Dis Child* 1966; 41:613-635.
8. Whittle MW, Evans M: Instrument for measuring the Cobb angle in scoliosis. *Lancet* 1979; i:414.
9. Lawton JO, Dickson RA: The experimental basis of idiopathic scoliosis. *Clin Orthop* 1986; 210:9-17.

(Calendar continued)

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October 11-13

The Eastern Orthopaedic Association will conduct a hands-on course entitled, "**Arthroscopy After the Knee,**" at the Homestead in **Hot Springs, Virginia**. This course will include arthroscopy of the shoulder, wrist, and ankle. Accommodations for 100 registrants only will be available. For further information, contact Elizabeth F. Capella, CMA-A, Suite 3F, 301 South 8th Street, Philadelphia, PA 19106-4072, or call (215) 923-9173.

October 14-18

The **Eighteenth Annual Meeting of the Eastern Orthopaedic Association** will be held at the Homestead in **Hot Springs, Virginia**. This meeting is open to members and non-members sponsored by members. For further information, contact Elizabeth F. Capella, CMA-A, Suite 3F, 301 South 8th Street, Philadelphia, PA 19106-4072, or call (215) 923-9173.

October 30-November 3

"**Tutorial on Level II Craniosacral Technique**" will be held at the Kellogg Center for Continuing Education, Michigan State University, **East Lansing, Michigan**. The purpose of the program is to review in detail cra-

nial anatomy; to review specific corrective techniques for complex articular restrictions; to learn soft-tissue corrective techniques for membranous strain patterns; to discuss special problems including TMJ, pediatric problems, entrapment syndromes, functional anatomy, treatment procedures. For further information, contact Sandy Kilbourn, MSU, East Fee Hall, East Lansing, MI 48824-1316.

November 20-22

"**Tutorial on Manual Medicine Techniques Applied to the Extremities**" will be held at the Kellogg Center for Continuing Education, Michigan State University, **East Lansing, Michigan**. This three-day intensive course will include the principles of manual medicine as they are applied to the upper and lower extremities. Emphasis will be placed on diagnosis and manipulative management of extremity problems. For further information, contact Sandy Kilbourn, Michigan State University, East Fee Hall, East Lansing, MI 48824-1316.

December 5

The **Third Annual Orthopedic Conference** sponsored by Michigan State University College of Osteopathic Medicine will be held at the Kellogg Center for Continuing Education in **East Lansing, Michigan**. For further information, contact Sandy Kilbourn,

Michigan State University, East Fee Hall, East Lansing, MI 48824-1316.

December 5-12

Post-RSNA Imaging at Sea will be held on a **Caribbean Cruise**. Victor Haughton, MD, Professor of Radiology Medical College of Wisconsin, will present, "Topics in Computed Tomography and Magnetic Resonance Imaging" through lectures, seminars, and case discussions. The program is sponsored through the Department of Radiology, Nu Med Medical and will be of value to practicing radiologists, neurosurgeons, neurologists, orthopedists, and interested clinicians. For further information, contact Medical Seminars International, Inc., 21915 Roscoe Boulevard, Suite 222, Canoga Park, CA 91304, or call (818) 340-0580, Extension 280.

December 14-18

"**Principles of Manual Medicine**" will be held at the Kellogg Center for Continuing Education, Michigan State University, **East Lansing, Michigan**. This course is a combination of didactic lectures and hands-on experience sufficient to understand the principles involved in the diagnosis and treatment of musculoskeletal disorders amenable to manual medicine methods. For further information, contact Sandy Kilbourn, Michigan State University, East Fee Hall, East Lansing, MI 48824-1316.

SPINAL GROWTH.

Dickson RA, Deacon P.

Journal of Bone and Joint Surgery; 69B: 690-692, 1987.

This annotation on spinal growth pointed out that the spine does not stop growing until the middle of the third decade. Progressive curves can therefore deteriorate until that time because the spine is still growing rather than any other soft tissue factor or pregnancy being in any way related to curve progression. The iliac crest apophysis and the vertebral apophyses were also shown to be unrelated to spinal growth.

The Journal of Bone and Joint Surgery

EDITORIAL

REFERENCES

You must have read articles which begin like this: "Colles' fracture is common in middle-aged women (Tom 1980; Dick 1920; Harry 1930) and many different methods of treatment have been recommended (Pfefferkorn 1951; Smith 1952; Pfefferkorn and Smith 1953; Smith and Pfefferkorn 1954), though with conflicting results (Klippel and Feil 1922; Gilbert and Sullivan 1953; Harrington and Rod 1968)". Perhaps you find that bracketed names interrupt your reading and prefer this: "Colles' fracture is common in middle-aged women,^{21,7,15} and many different methods of treatment have been recommended,^{5,1,18,9,} though with conflicting results^{10,4,25}". This is easier to read though, without repeated turning of pages, you have no idea when they were published, nor if the numbered authors are noteworthies or nobodies. Each system has advantages, but their relative merits need not concern us here – there are more important issues: multiplicity and accuracy.

It has been said that numbering promotes multiplicity, but the real question is, did we need any references at all? Surely we would accept that Colles' fracture is common in middle-aged women and that treatment varies, without needing the reassurance of looking up all those papers. Indeed we may wonder if the author himself has looked them all up or whether he has copied some from a previous author's list, hoping to gain

reputation by proxy, so to speak. The true story is told of an author who deliberately included among his references one which was totally fictitious and, until he confessed, enjoyed finding it in the lists of later authors.

As for accuracy, this is one of the hallmarks of good scientific publications, but you may be surprised to learn that a considerable proportion of the papers we receive contain errors such that the reader would fail to find the listed articles. This is why we try very hard to check every reference listed back to its source. Occasionally we fail and have to ask the author for a photocopy (if this is not forthcoming it is hard to resist the suspicion that it was a "proxy").

Please do not imagine that this editorial aims to deter authors from citing articles – far from it; some will be needed to convince us that the work has been adequately researched, to enable techniques to be cited rather than described, or to compare the findings with those of others. References are like insulin; when appropriate both are essential, but in excess they induce coma, and impurities may be disastrous. Our object is to persuade authors to use references with discrimination, including only those which have real point, and to record these select few with meticulous pedantry. Journals, librarians, readers and future workers will heap blessings upon the head of the author who follows these two precepts; reviewers and editors may even go so far as to accept his paper for publication.

ANNOTATION

SPINAL GROWTH

A constant and worrying factor in children's orthopaedic surgery is growth. This occurs in three dimensions at the same time, at different rates in different parts of the body, for two decades or more with increased rates in utero and during adolescence. Growth may benefit malunited fractures by allowing remodelling from wide limits of acceptability, but on the other hand it may militate against improvement, particularly in deformity of early onset. The notorious tendency of club foot deformity to relapse, despite treatment, is one example of the unfortunate effect of growth; but for unpredictability during development the most difficult deformity is scoliosis.

Both conservative treatment and spinal fusion operations are used to counter the effects of growth; when this has ceased progression of the deformity is no longer important. Yet despite much effort directed to the amelioration of the unfavourable effect of growth, little attention has been paid to the facts of spinal growth. Nonetheless, over the years, many important points have been clarified; knowledge of these is crucial to an understanding of the behaviour of spinal deformities.

The assessment of spinal maturity. In the belief that spinal deformities cease to progress after the attainment of skeletal maturity, much work has been directed towards the accurate identification of this time. Risser, a pioneer in scoliosis surgery, demonstrated that skeletal maturity could be assessed from the ossification of the apophysis of the iliac crest (Risser and Ferguson 1936; Risser 1958). Ossification of the apophysis first appears in the region of the anterior superior iliac spine and then spreads posteriorly and medially until the whole apophysis is ossified. During the next year the apophysis fuses to the ilium. This process can be quantified by considering the iliac crest in quarters. Scoliosis surgeons, eager for some measurement of maturity, readily adopted this system, referring to their patient's status as, say, Risser 4. In 1936 Risser and Ferguson reported on almost 300 children with scoliosis and observed that curve progression, which had formerly averaged 1° per month, stopped when the ossific nucleus reached the posterior superior iliac spine. This occurred at an average age of 14 years for girls and 15.5 for boys.

James (1954) confirmed this end point when he studied his patients with idiopathic scoliosis and noted that apophyseal fusion occurred synchronously in the iliac crest and the vertebrae (Zaoussis and James 1958). Other authors supported the use of these apophyses to assess maturity (Clarisse 1974; Heine and Reher 1975); the method was quickly adopted and is now widely used. Follow-up, however, did not extend far into the post-mature phase and failed to provide direct evidence of the

relationship between apophyseal fusion and the growth of the spine itself.

Broadhurst (1864) had previously come to quite different conclusions, observing that "distortion of the spine is never stationary but always advances slowly", while Adams (1865), in his wonderfully informative essay on scoliosis, stated "it is impossible to estimate too highly the importance of studying the natural progress of any disease as it pursues its course when not interfered with by medical art". He observed that the curves in his scoliosis patients progressed after their maturity and could increase at any age. Calvo (1957) observed that spinal growth does not stop when the iliac apophyses had completed their ossification. Tupman's important and detailed study of limb and spine growth in normal children (1962) was recognised by the award of the Robert Jones Gold Medal. He measured tibial and femoral growth, and showed that the tibia and femur stopped growing in girls at the mean ages of 13.7 and 14.2 years respectively and in boys at 15.5 and 15.4 years respectively, while growth of the trunk was not finished in either sex by the age of 16 years, the end point of his study.

More evidence has accumulated in favour of the progression of curves beyond maturity. Ponseti and Friedman (1950) reported that curves progressed about 1° per year in adulthood, while another report from Iowa demonstrated that the average increase in curve after maturity was 15° , and that curves of 60° or more were particularly likely to deteriorate (Collis and Ponseti 1969). Dickson and Leatherman (1976) found 10 cases of idiopathic scoliosis which had radiographs taken after the attainment of general skeletal maturity and also some years later. In these cases the initial mean curve of 44° had progressed to 60° after a mean interval of six years. One curve had increased from 40° at the age of 20 years to 95° at the age of 33 years. These observations were confirmed by others and it was noted that adults could lose as much as 24 cm in height as a result of progression of spinal deformity (Stagnara, Gonan and Fauchet 1984). It was suggested by some that this progression was due to pregnancy or some mysterious soft-tissue factor, although little evidence was provided (Hassan and Bjerkreim 1983; Weinstein and Ponseti 1983).

Vertebral growth. Vertebral growth occurs in a similar fashion as growth in the long bones (Haas 1939). Increase in length and change in shape takes place as a result of activity in the physal plates, there being no interstitial growth. In the vertebrae, there being no bony epiphyses, growth cartilage lies between the bone of the vertebral body and the tissues of the intervertebral disc. Confusion between the roles of the vertebral ring apophysis and the

physeal plate persist despite careful growth studies (Bick and Copel 1950, 1951).

The vertebral ring apophyses are merely the peripherally ossified part of the cartilaginous vertebral plate, at the attachment of ligament or periosteum beyond the perichondrial ring (Figs 1 and 2). They fuse with the vertebral body long before the end of spinal

about the attainment of general skeletal maturity, they bear no relationship to spinal growth or its cessation. Of course, because the vertebral growth plates are still present until an average age of 25 years, this does not imply that significant longitudinal growth continues to occur until this time. Indeed, it has been clearly demonstrated that vertebral bodies are half the adult size



Fig. 1



Fig. 2

Figure 1 – Lateral radiograph of thoracic vertebrae showing the step-like recesses in the upper and lower margins of the ossific vertebral body which are due to the cartilage of the vertebral epiphyseal plates. Figure 2 – Lateral radiograph showing the vertebral apophyses: regions of ossification at the sites of ligamentous insertion. Note that these are outside the vertebral epiphyses.

growth. Bick clearly showed that the ring apophysis takes no part in longitudinal growth, nor does its ossification have any relationship to this growth. He observed that calcification, and later ossification, in the ring apophysis lies outside the plane of the physal growth plate and that apophyseal fusion occurs over several years with different timing in different regions of the spine. If the term "traction apophysis" were used, as suggested by Bick, then confusion with the physal growth plate would not arise. These observations have been confirmed (Larsen and Nordentoft 1962) and a histological study of the cartilaginous end-plates from birth to 73 years of age has demonstrated that growth cartilage is present but decreasing in width until the patient's age is well into the twenties (Bernick and Cailliet 1982). Since the vertebral bodies have no secondary centres of ossification as is the case in long bones, there is no epiphysis to fuse with the diaphysis and so demonstrate the obliteration of the growth cartilage which is seen at the time of skeletal maturity elsewhere in the skeleton.

Therefore while the status of ossification of the iliac crest and vertebral ring apophyses may say something

by the age of two years, with little longitudinal growth occurring after the age of 10 years (Haas 1939; Bick and Copel 1950; Larsen and Nordentoft 1962; Bernick and Cailliet 1982). Nonetheless, while the end-plates are open, time for change in shape is available. This may be of little importance in the symmetrical spine, but may be crucial in the presence of a structural scoliosis, which is subject to enormous asymmetrical forces (Dickson et al. 1984). It would therefore be unwise to postulate hormonal factors or pregnancy as causes of curve progression in early adult life when the spine has not yet stopped growing.

Although the time of spinal maturity can thus be estimated no more accurately than "during the mid-twenties", there is little point in effort at its exact determination.

Assessment of growth. What is much more important is to discover how scoliotic children grow, particularly during adolescence when increased growth velocity heralds the danger of curve progression. While some information can be gleaned in this respect from the Risser scale, there are many more accurate methods available. Chronological age, although always recorded, is a notoriously

inaccurate measure of true biological age.

Fifty years ago in Cleveland growth studies on normal children were performed and, in particular, the development of the bones of the left hand and wrist were estimated radiographically (Todd 1937). This important work formed the basis of the Gruelich and Pyle (1959) atlas, against whose standards the radiographic appearance of the hand and wrist could be compared. Measurement of biological age therefore became much more accurate but there were two principal drawbacks. The upper-class Cleveland children who formed the basis of the atlas still appear to be among the most advanced on record and, secondly, most of the age intervals are one year, though some are six months. If a child does not fulfil all the criteria for a certain age, then biological age is assigned to the previous year; there is therefore a consistent tendency to under-rate the biological age.

More up-to-date standards are now available. The TW2 bone age (Tanner et al. 1983), although a more time-consuming method, is more accurate. There is currently no objection to the taking of a radiograph of the hand and wrist in either scoliosis patients or normal children. If the natural history of scoliosis in relation to biological growth is to be elucidated then all such patients should have this simple test performed at every visit, so that the longitudinal manner in which that child is going through the important growth phase of adolescence can be accurately assessed. Similarly, there are standards for standing height, sitting height and puberty ratings (Tanner and Whitehouse 1975) and these are depicted on centile charts, without which no scoliosis record should be considered complete. While hand and wrist radiographs are safe and height measurement is harmless, the same cannot be said for repeated x-ray dosage of the pelvis of growing children, even when a low-dose technique is used (Adran et al. 1980).

Conclusions. Although physcal growth occurs in the same manner in the spine as elsewhere in the skeleton, and is subject to the same governing factors, there is no completely satisfactory method of determining spinal maturity in children and adolescents; the status of ossification of the iliac crest and of the vertebral ring apophyses is irrelevant.

The best method of assessing maturity is an amalgam of bone age, height measurements and puberty ratings, and it is clear that on average the spine grows for ten years longer than the lower limbs. The effect on simple longitudinal growth is marginal; but the effect on the progress of scoliosis should not be underestimated.

ROBERT A. DICKSON
PHILIP DEACON

REFERENCES

- Adams W. *Lectures on the pathology and treatment of lateral and other forms of curvature of the spine*. London: Churchill, 1865.
- Adran GM, Coates R, Dickson RA, Dixon-Brown A, Harding FM. Assessment of scoliosis in children: low-dose radiographic technique. *Br J Radiol* 1980;53:146-7.
- Bernick S, Cailliet R. Vertebral end-plate changes with aging of human vertebrae. *Spine* 1982;7:97-102.
- Bick EM, Copel JW. Longitudinal growth of the human vertebra: a contribution to human osteogeny. *J Bone Joint Surg [Am]* 1950;32-A:803-14.
- Bick EM, Copel JW. The ring apophysis of the human vertebra. Contribution to human osteogeny. II. *J Bone Joint Surg [Am]* 1951;33-A:783-7.
- Broadhurst BE. *On lateral curvature of the spine*. 2nd ed. London: John Churchill & Sons, 1864.
- Calvo JJ. Observations on the growth of the female adolescent spine and its relation to scoliosis. *Clin Orthop* 1957;10:40-7.
- Clarisse P. *Prognostic évolutif des scolioses idiopathiques mineures de 10° à 29° en période de croissance*. Doctoral thesis. Lyons: Claude Bernard, 1974.
- Collis DK, Ponseti IV. Long-term follow-up of patients with idiopathic scoliosis not treated surgically. *J Bone Joint Surg [Am]* 1969;51-A:425-45.
- Dickson RA, Leatherman KD. Spinal deformity in adults: changing concepts. *J Bone Joint Surg [Am]* 1976;58-A:729.
- Dickson RA, Lawton JO, Archer IA, Butt WP. The pathogenesis of idiopathic scoliosis: biplanar spinal asymmetry. *J Bone Joint Surg [Br]* 1984;66-B:8-15.
- Gruelich WW, Pyle SI. *Radiographic atlas of skeletal development of the hand and wrist*. 2nd ed. Stanford: Stanford University Press; and London: Oxford University Press, 1959.
- Haas SL. Growth in length of vertebrae. *Arch Surg* 1939;38:245-9.
- Hassan I, Bjerkreim I. Progression in idiopathic scoliosis after conservative treatment. *Acta Orthop Scand* 1983;54:88-90.
- Heine J, Reher H. Die Progredienz der unbehandelten Skoliose nach Poliomyelitis bis Wachstumsabschluss. *Z Orthop* 1975;113:876-9. (Eng. Abstr.)
- James JIP. Idiopathic scoliosis: the prognosis, diagnosis and operative indications related to curve patterns and the age at onset. *J Bone Joint Surg [Br]* 1954;36-B:36-49.
- Larsen EH, Nordentoft EL. Growth of the epiphyses and vertebra. *Acta Orthop Scand* 1962;32:210-7.
- Ponseti IV, Friedman B. Prognosis in idiopathic scoliosis. *J Bone Joint Surg [Am]* 1950;32-A:381-95.
- Risser JC. The iliac apophysis: an invaluable sign in the management of scoliosis. *Clin Orthop* 1958;11:111-9.
- Risser JC, Ferguson AB. Scoliosis: its prognosis. *J Bone Joint Surg* 1936;18:667-70.
- Stagnara P, Gonan G-P, Fauchet P. Surgical treatment of idiopathic rigid lumbar scoliosis in the adult. In: Dickson RA, Bradford DS, eds. *Management of spinal deformities*. Butterworths International Medical Reviews: Orthopaedics 2. London etc: Butterworths, 1984;303-21.
- Tanner JM, Whitehouse RH. *Height standard chart*. Castlemead: Creaseys, 1975.
- Tanner JM, Whitehouse RH, Cameron N, Marshall WA, Healy MJR, Goldstein H. Assessment of skeletal maturity and prediction of adult height (TW2 method). 2nd ed. London: Academic Press, 1983.
- Todd TW. *Atlas of skeletal maturation*, Part I: Hand. London: Henry Kimpton, 1937;137-203.
- Tupman GS. A study of bone growth in normal children and its relationship to skeletal maturation. *J Bone Joint Surg [Br]* 1962;44-B:42-67.
- Weinstein SL, Ponseti IV. Curve progression in idiopathic scoliosis. *J Bone Joint Surg [Am]* 1983;65-A:447-55.
- Zaoussis AL, James JIP. The iliac apophysis and the evolution of curves in scoliosis. *J Bone Joint Surg [Br]* 1958;40-B:442-53.

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THE AETIOLOGY OF SPINAL DEFORMITIES.

Dickson RA.

Lancet; 1: 1151-1155, 1988.

This paper extended the concept that abnormalities of lateral profile are primarily pathogenetic in spinal deformities of the non-idiopathic variety and thus all spinal deformities could be explicable in simple biological and biomechanical terms.

Dogma Disputed

THE AETIOLOGY OF SPINAL DEFORMITIES

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Summary There are two types of spinal deformity, lordosis and kyphosis, and they are mutually exclusive at the same site. Lordosis is rotationally unstable and buckles to the side with growth and spinal flexion, producing scoliosis and changes in transverse plane geometry as secondary phenomena. Kyphosis is a uniplanar deformity arising behind the axis of spinal column rotation and it does not buckle. Spinal balance in the sagittal plane is delicate and in the normal child during adolescence both idiopathic scoliosis and idiopathic kyphosis can easily develop. The development and progression of spinal deformities can be explained in biological and mechanical terms. Any condition in which the critical load to the spine is reduced will favour the production and progression of a spinal deformity. Neuromuscular factors in idiopathic scoliosis are additive and not causative.

INTRODUCTION

ON the unlikely assumption that teenage girls with idiopathic scoliosis have sub-clinical muscular dystrophy or poliomyelitis, workers have long searched for a neuromuscular causative factor. Since before Lericq and Le Coeur¹ first demonstrated by electromyography action potential asymmetry between the paravertebral musculature on each side of an idiopathic scoliosis, brain,² eyes,³ ears,⁴ spinal cord,⁵ nerve roots,⁶ muscles,⁷ collagen,⁸ rib heads,⁹ costo-transverse ligaments,¹⁰ and even platelets¹¹ have been implicated in idiopathic scoliosis to the point where one asks: why do such patients appear normal, with the exception of their spinal deformity?¹² Fortunately, the situation is not so complex or horrific as such research suggests, and both the cause and behaviour of spinal deformities can be explained in simple mechanical and biological terms.

ALL STRUCTURAL SCOLIOSES ARE LORDOTIC

A postero-anterior (PA) radiograph of a patient with structural scoliosis demonstrates a lateral curvature of the spine and rotation (fig 1). The posterior elements of the spine are rotated into the concavity and the anterior bodies

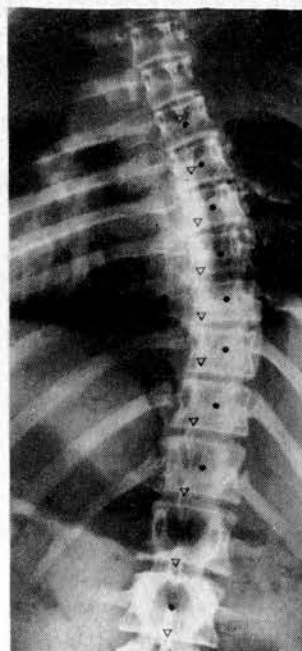


Fig 1—PA radiograph of an idiopathic thoracic scoliosis.

▽ = spinous processes, ● = vertebral bodies.

are rotated into the convexity of the curve. The back of the curved spine is therefore shorter than the front.¹³ Hence the deformity is lordotic, and since this pattern of rotation accompanies every structural scoliosis at any site in the spine there is no such deformity as kyphoscoliosis, a much used term. That all structural scolioses are lordotic was known 120 years ago,¹⁴ before radiographs of the spine misled us into believing that the apparent lateral curvature was important in the development of the three-dimensional deformity. However, until lately this important notion has only received sporadic attention.^{13,15}

Since the PA radiograph of the patient encompasses rotation, it is an oblique view of the deformity and thus a lateral view of the patient, which shows another oblique view of the same deformity. Thus, one receives a spurious impression of kyphosis because the spinal column appears to point backwards. To take a true lateral projection the X-ray beam must be turned 90° to the rotation of the vertebra at the apex of the deformity, and when this true planar view is obtained the deformity proves to be consistently lordotic (fig 2a)—the degree of lordosis being proportional to the degree of scoliosis as seen on the PA projection of the patient.¹⁶

D. G. JAGELMAN AND OTHERS: REFERENCES

- Hoffman DC, Goligher JC. Polyposis of the stomach and small intestine in association with familial polyposis coli. *Br J Surg* 1971; **58**: 126-28.
- Jones TR, Nance FC. Periapillary malignancy in Gardner's syndrome. *Ann Surg* 1977; **185**: 565-73.
- Watanabe H, Enjoji M, Yao T, Ohsato K. Gastric lesions in familial adenomatous polyposis coli: their incidence and histologic analysis. *Hum Pathol* 1978; **9**: 269-82.
- Harned RK, Williams SM. Familial polyposis coli and periampillary malignancy. *Dis Colon Rectum* 1982; **25**: 227-29.
- Sener SF, Miller HH, DeCossé JJ. The spectrum of polyposis. *Surg Gynecol Obstet* 1984; **159**: 525-32.
- Jarvinen HJ, Nyberg M, Peltokallio P. Biliary involvement in familial adenomatous polyposis coli. *Dis Colon Rectum* 1983; **26**: 525-28.
- Coffey RJ, Knight CD Jr, van Heerden JA, Weiland LH. Gastric adenocarcinoma complicating Gardner's syndrome in a North American woman. *Gastroenterology* 1985; **88**: 1263-66.
- Silverberg E, Lubera J. Cancer statistics, 1987. *CA* 1987; **37**: 2-20.
- Longmire WP Jr. Periapillary tumours. *J R Coll Surg Edin* 1973; **18**: 131-36.
- Hauser G. Über polyposis intestinalis adenomatosa und deren Beziehungen zur Krebsentwicklung. *Arch Klin Med* 1895; **55**: 429-48.
- Sarre RG, Frost AG, Jagelman DG, Petras RE, Sivak MV, McGannon E. Gastric and duodenal polyps in familial adenomatous polyposis: a prospective study of the nature and prevalence of upper GI polyps. *Gut* 1987; **28**: 306-14.
- Kurtz RC, Sternberg SS, Miller HH, DeCossé JJ. Upper gastrointestinal neoplasia in familial polyposis. *Dig Dis Sci* 1987; **32**: 459-65.
- Kozuka S, Tsubone M, Yamaguchi A, Hachisuka K. Adenomatous residue in cancerous papilla of Vater. *Gut* 1981; **22**: 1021-34.
- Snow AD, Altmann GG. Morphometric study of the rat duodenal epithelium during initial six months of 1,2-dimethylhydrazine carcinogenesis. *Cancer Res* 1983; **43**: 4838-49.
- Ward JM. Morphogenesis of chemically induced neoplasms of the colon and small intestine in rats. *Lab Invest* 1974; **30**: 112-20.
- Bissey HJR, Fyers AA, Ritchie SM, Thomson JPS. The rectum in adenomatous polyposis: the St. Mark's policy. *Br J Surg* 1985; **72**: 29-31 (suppl).

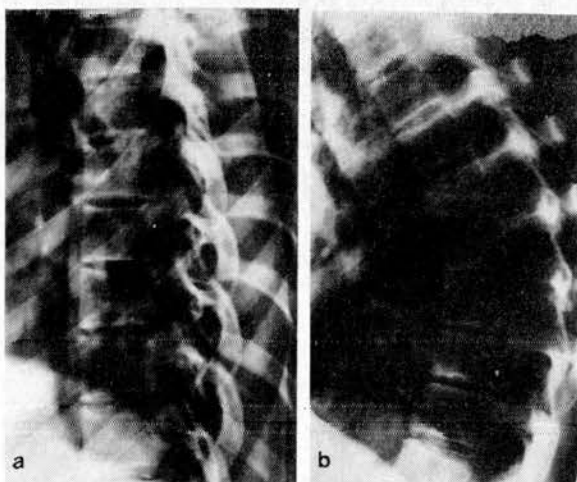


Fig 2—*a*, true lateral radiograph of the apex of the curve shown in fig 1; *b*, lateral radiograph of the thoracic spine in Scheuermann's kyphosis.

Thus, although the lateral view of the patient shows a spinal column that appears convex backwards, it is the anterior aspect of the spine that points posteriorly since the lordotic region has rotated away from the sagittal plane. In the thoracic region, which is kyphotic in the normal person, a substantial departure from the normal sagittal shape has clearly developed (fig 2a) and true lateral projections of deformities lower down in the spine show lordosis where the spine should be flat (thoraco-lumbar region) and increased lordosis where some lordosis is normal (lumbar region). It is, however, the thoracic region where these changes are most obvious. True lateral projections of the apex of an idiopathic thoracic scoliosis show that this lordosis is accommodated at bone level, the vertebrae being wedged, with a greater anterior than posterior height and any Schmorl node formation or end-plate irregularity being posterior in the growth plates.¹⁵

IDIOPATHIC SCOLIOSIS VERSUS SCHEUERMANN'S DISEASE

Lateral radiographs of Scheuermann's kyphosis, the other idiopathic spinal deformity during immaturity, show the same changes as are found in idiopathic scoliosis, but in the opposite direction (fig 2b): the wedged vertebrae have a much shorter anterior than posterior height and any Schmorl-node formation or end-plate irregularity is anterior in the growth plate.¹⁷ As in idiopathic scoliosis, these children are normal on physical examination in every other respect and present as a result of their deformity, although they often have a dull ache. Moreover, the two types of deformity occur at the same level in the thoracic spine (T8, T9) and are common, with much the same community prevalence rates. Idiopathic scoliosis of clinical significance affects females more than males, whereas Scheuermann's disease tends to affect males; but parallels can be drawn here too. The development of these deformities provides the link.

The sagittal plane differences in idiopathic thoracic scoliosis and Scheuermann's disease appear to be quite striking, but they are not, which illustrates the precarious balance in the sagittal plane. In late childhood, the middle eight thoracic vertebrae are evenly kyphotic (before the early adolescent flattening process) by about 3° in each, making a total thoracic kyphosis of about 24°. The first two and last

two thoracic vertebrae are in the flatter areas of the kyphosis, conjoining the cervical and lumbar lordoses. Scheuermann's disease is diagnosed by the radiographic appearance of 5° of wedging of each of three consecutive vertebrae.¹⁸ Therefore, the sagittal profile has only to alter by 2° in each vertebra for Scheuermann's disease to be diagnosed and by only just over 3° in the opposite direction for the thoracic spine to be truly lordotic. Such changes that are slight during slow or steady growth rates become much greater during rapid growth. Thus during the adolescent growth spurt, when the spine is growing at its fastest since birth (and in three dimensions at the same time) the effect of small changes in the sagittal profile is at its greatest in producing progressive spinal deformities. Genetic factors, too, become important at this stage of development. The spinal profile, like every other aspect of body shape, has strong genetic expression, round backs coming from round-backed families and flat backs coming from flat-backed families.^{19,20} It is not therefore surprising to find this familial trend in both idiopathic scoliosis and Scheuermann's disease,¹⁸ but to rename idiopathic scoliosis genetic or familial scoliosis²¹ would be ill-advised without an understanding of the three-dimensional nature of its pathogenesis.

Body shape changes with growth,²² and even thereafter, as does the spinal profile. In late childhood and early adolescence the normal thoracic kyphosis reduces considerably at the same age in both sexes.²³ In girls this flattening process corresponds with peak adolescent growth velocity, when boys on average are still some two years behind biologically. In some girls this combined phase of spinal growth acceleration and flattening of the thoracic kyphosis predisposes to the normal sagittal profile reversing into a lordosis (idiopathic scoliosis). Meanwhile boys, who have not yet entered their phase of peak adolescent growth velocity, are more protected than girls. Two years later, however, when the normal thoracic kyphosis is regained, boys are at their peak adolescent growth velocity and this in turn predisposes in some to an increased thoracic kyphosis (Scheuermann's disease).

MECHANICAL FACTORS

Superimposed upon these growth changes mechanical factors become crucial. The axis of spinal column rotation passes through the spine from foramen magnum to sacrum behind the areas of cervical and lumbar lordoses and in front of the thoracic kyphosis. On forward flexion, areas of lordosis are vulnerable to buckling since they lie anterior to the axis of rotation. The cervical and lumbar regions, however, are well protected against buckling:²⁴ they have powerful posterior muscles and ligaments, whose symmetrical payout assists greatly in maintaining the integrity of these lordoses. Moreover, the increased disc height in the cervical and lumbar regions confers a considerable amount of flexion before the column would come under compression and thus buckle. In addition, both cervical and lumbar vertebral bodies are much broader from side to side than they are from front to back and thus resemble prisms, with their bases anteriorly. When such a prismatic configuration is flexed the reduced cross-sectional moment of area tends to resist buckling.

In the thoracic region, however, flexion is restricted by the narrow disc height, posterior spinal muscle support is much less than in the cervical and lumbar regions, and the vertebrae are heart-shaped, resembling a prism with its apex

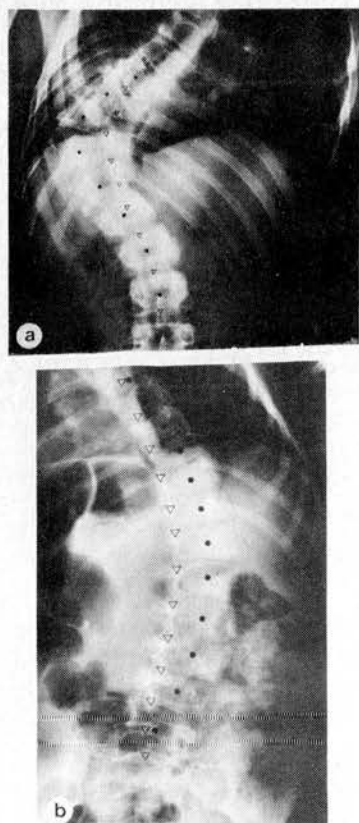


Fig 3—PA radiographs of (a) left thoracic curve in Von Recklinghausen's disease and (b) paralytic lumbar scoliosis associated with spinal muscular atrophy.

anteriorly—a rotationally unstable configuration.²⁴ The thoracic spinal column is therefore protected by the presence of a kyphosis such that its vulnerable part lies behind the axis of rotation and is thus under tension and not compression. As an area of lordosis develops, however, the vulnerable column is shifted to the wrong side of the axis of rotation, comes under compression, and buckles to the side with growth. There is too much bony material to be accommodated and particularly when the column is flexed it must go somewhere.¹⁵ The forward bend test is used by scoliosis surgeons and those screening for the condition because during flexion the rotational prominence is increased, or in small curves it becomes apparent when it was not discernible in the erect position.^{14,15}

Biological tissue moves in a hysteresis fashion, not quite returning to its original position before the next cycle of flexion in the activities of daily living supervenes. In the curved spine the vertebral growth plates now confer a secondary mechanical disadvantage by gradually growing more slowly on the compression side than on the tension side. The essential lordosis thus moves progressively away from the sagittal plane towards the coronal plane producing a lateral curvature of the spine, or scoliosis, as a secondary phenomenon. Meanwhile, the degree of lordosis seen on a true lateral projection, although it is still increasing, is much less than the degree of progression observed in Scheuermann's disease. If the mechanics were favourable and the lordosis remained in the sagittal plane, posterior growth plate compression would stay at maximum and a pure lordosis, akin to the kyphosis of Scheuermann's disease, would be produced. However, since the lordosis

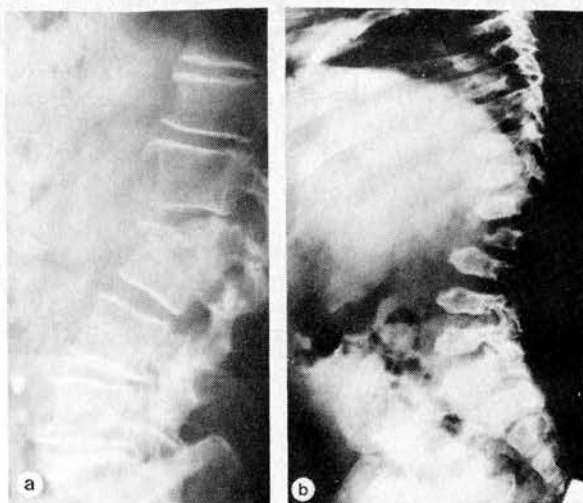


Fig 4—Lateral radiographs of (a) lumbar spine showing kyphosis produced by osteomyelitis and (b) thoraco-lumbar spine in Morquio's syndrome.

In b there is a kyphosis with an apical bullet-shaped vertebra; the anterior column has not developed properly.

moves away from the sagittal plane, posterior growth plate pressure diminishes as a true lateral curvature develops, and thus the degree of lordosis and of scoliosis are significantly correlated.¹⁶

At the same time the geometry in the transverse plane is also altering. Changes in the length and thickness of pedicle are secondary. As the apical region of the curve moves to the side the convex pedicle of the apical vertebrae becomes shorter and thicker, being under compression, whereas the concave pedicle becomes longer and attenuated.²⁵ Simultaneously, bone flow takes place, with the vertebral body tending to drift back towards the mid-line. These changes in the transverse plane become progressively less obvious in vertebrae away from the apical region of the curve, until above and below the five apical vertebrae a symmetrical vertebra is encountered. Above these symmetrical vertebrae, in the areas of asymmetric kyphosis that are compensatory to the apical region of buckled lordosis, the transverse plane changes occur in the opposite direction (ie, the convex pedicle becomes longer and attenuated and the concave pedicle becomes shorter and thicker), until the vertebrae again become symmetrical at the level of the first parallel vertebrae.

CLINICAL CONSEQUENCES

There are therefore two primordial types of spinal deformity, lordosis (which buckles to the side to produce the secondary deformity of scoliosis) and kyphosis (a uniplanar rotationally stable deformity that is in the sagittal plane only).²⁶ The kyphotic process, however, is seldom completely symmetrical in the coronal plane and a slight scoliosis is often seen on the PA radiograph.¹⁷ The spinous processes are, however, either not rotated or mildly rotated into the convexity of this scoliotic curve, indicating that the deviation is a kyphosis that is asymmetric and is not a buckled lordosis. These scolioses, lying behind the axis of spinal rotation, are never progressive.

How a column fails in engineering terms and the subsequent progression of that failure are well known, as are

the laws that govern them.²⁶ There are two types of column failure—brittle and plastic. The former is simple angular collapse due to material failure under either compression or tension, whereas the latter is the phenomenon of beam buckling, which need not encompass any true material failure but is the result of rotational instability and involves all three dimensions. The variables that govern buckling of a beam can be simplified into Euler's law— $F(\text{crit}) = C(EI/L^2)$, where $F(\text{crit})$ is the critical load, C is the end conditions, E is the modulus of elasticity, I is the cross-section moment of inertia of the buckling plane, and L is the beam length. Structural scolioses of the non-idiopathic variety can be explained by Euler's law. Factors that reduce the critical load favour both buckling and its progression, and in the spine this can occur in the soft tissue, bone, or muscle. The heritable connective tissue disorders of Marfan's syndrome, homocystinuria, and Ehlers-Danlos syndrome are associated with a high prevalence rate of structural scoliosis and an increased progression potential. In these disorders the radiographic deformity (a lateral curvature with rotation) is, however, indistinguishable from idiopathic scoliosis, the diagnostic features of these conditions being identified by physical examination. The critical load has been reduced at the soft tissue level.

The deformities seen in von Recklinghausen's disease, osteogenesis imperfecta, the mucopolysaccharidoses, the skeletal dysplasias, and metabolic bone disorders have the common denominator of weak bone, thus reducing the critical load at bone level. For example, in von Recklinghausen's disease the prevalence rate of idiopathic-type deformities that measure 20° is 150 times greater than that of its true idiopathic counterpart (fig 3a). Moreover, their progression potential is well known. Although the normal lumbar lordosis has the symmetrical support of its posterior paravertebral musculature, asymmetric muscle failure due to paralysis rapidly allows the lordosis to buckle (fig 3b). Thus all structural scolioses are the same three-dimensional deformities subject to the same mechanical laws, but biological factors reduce their ability to resist buckling. Clearly the child who is otherwise normal is best able to resist buckling and thus has the lowest prevalence rate and progression potential.

In the idiopathic form of structural scoliosis, however, there is no clear evidence of true material failure. Euler's law also operates in other respects—in particular the length of the column (the longer it is the more likely it is to buckle) and the end conditions (fixed or otherwise, the former favouring buckling) are important. Children with idiopathic scoliosis are marginally but significantly taller than those with straight backs,²⁷ and there is no structural scoliosis where the ends of the beam are not fixed by the head and pelvis pointing forwards.

The simple angular collapse of kyphosis implies true material failure. This collapse occurs by failure of the anterior column under compression in conditions such as trauma, tumour, infection, skeletal dysplasia, or posterior element failure under tension, as happens in trauma, laminectomy, and some neuromuscular conditions (fig 4). Thus all spinal deformities (lordoses or kyphoses), can be explained in mechanical and biological terms. There is no need to search for some factor that differentiates a 100° curve from a 20° curve in respect of progression potential since the 100° curve will progress more rapidly because beam failure is exponential—the bigger the deformity, the more likely it is to progress.²⁶ The question should therefore be: why did the

100° deformity arise in the first place? Here, neuromuscular factors may be important. Idiopathic scoliosis in infancy resolves in 90% of cases but progresses in about 10%, and progression is associated with low birthweight, hypotonic, floppy babies with low neurological development scores.²⁸ Moreover, neuropathological changes have been seen at necropsy.²⁹ In the initial stages in infants, however, the radiographic appearances of resolving and progressing curves are the same until increased rotation (as evidenced by the angle of the ribs) indicates the progressive form. It is the imperfection of the delicate postural control mechanisms of the spine that favours progression, by way of Euler's law, rather than such imperfection being the causative factor.

In adolescent children with idiopathic scoliosis, too, there is evidence that postural and equilibrium dysfunction is not uncommon in the progressive form of the disorder,^{3,4} although abnormal electro-encephalography has been refuted² and electrophysiological and muscle histological changes are secondary to the presence of a spinal curvature.^{30,31} Again, Euler's law has been obeyed. However, experimental work in animals suggesting that idiopathic scoliosis can be created by rib resection⁹ or division of the costotransverse ligament¹⁰ has been invalidated:³² such procedures damage the segmental arterial supply to the spinal cord and thus are models of paralytic scoliosis produced by direct nerve damage, which reduces the critical load at muscle level.

REFERENCES

1. Lericque, Le Coeur. Action potential asymmetry on two sides. 1951. Cited by Le Febvre J, Triboulet-Chassevant A, Missirlou MF. Electromyographic data in idiopathic scoliosis. *Arch Phys Med Rehabil* 1961; **42**: 710-11.
2. Robb JE, Conner AN, Stephenson JBP. Normal electroencephalograms in idiopathic scoliosis. *Acta Orthop Scand* 1986; **57**: 220-21.
3. Sahlstrand T, Petruson B. Postural effects on nystagmus response during caloric labyrinthine stimulation in patients with adolescent idiopathic scoliosis. II. An electronystagmographic study. *Acta Orthop Scand* 1979; **50**: 771-75.
4. Sahlstrand T, Petruson B. A study of labyrinthine function in patients with adolescent idiopathic scoliosis. I. An electronystagmographic study. *Acta Orthop Scand* 1979; **50**: 759-69.
5. De Salis J, Beguiristain JL, Canadell J. The production of experimental scoliosis by selective arterial ablation. *Int Orthop* 1980; **3**: 311-15.
6. Alexander MA, Bunch WH, Ebbesson SOE. Can experimental dorsal rhizotomy produce scoliosis? *J Bone Joint Surg* 1972; **54A**: 1509-13.
7. Zuk T. The role of spinal and abdominal muscles in the pathogenesis of scoliosis. *J Bone Joint Surg* 1962; **44B**: 102-05.
8. Bushell GR, Ghosh P, Taylor TKF. Collagen defect in idiopathic scoliosis. *Lancet* 1978; **ii**: 94-95.
9. Piggott H. Experimentally produced scoliosis in animals. In: Zorab PA, ed. Proceedings of a second symposium on scoliosis: causation. Edinburgh: E & S Livingstone 1968; 15-17.
10. Langenskiöld A, Michelsson JE. Experimental progressive scoliosis in the rabbit. *J Bone Joint Surg* 1961; **43B**: 116-20.
11. Yarom R, More R, Meyer S. Platelet and muscle abnormalities in idiopathic scoliosis. In: Warner JO, Mehta MH, eds. Proceedings of the seventh Phillip Zorab symposium: scoliosis prevention. New York: Praeger, 1983: 3-22.
12. Nachemson AL. Future research in scoliosis. In: Zorab PA, Siegler D, eds. Proceedings of the sixth symposium on scoliosis: scoliosis 1979. London: Academic Press, 1979: 263-70.
13. Roaf R. The basic anatomy of scoliosis. *J Bone Joint Surg* 1966; **48B**: 786-92.
14. Adams W. Lectures on the pathology and treatment of lateral and other forms of curvature of the spine. London: Churchill, 1865.
15. Dickson RA, Lawton JO, Archer IA, Butt WP. The pathogenesis of idiopathic scoliosis—bi-planar spinal asymmetry. *J Bone Joint Surg* 1984; **66B**: 8-15.
16. Deacon P, Flood BM, Dickson RA. Idiopathic scoliosis in three dimensions: a radiographic and morphometric analysis. *J Bone Joint Surg* 1984; **66B**: 509-12.
17. Deacon P, Berkin CR, Dickson RA. Combined idiopathic kyphosis and scoliosis: an analysis of the lateral spinal curvatures associated with Scheuermann's disease. *J Bone Joint Surg* 1985; **67B**: 189-92.
18. Sorenson KH. Scheuermann's juvenile kyphosis. Copenhagen: Munksgaard, 1964.
19. Delmas A. Types rachidiens de statique corporelle. *Rev Morphophysiol Hum* 1951; **4** (II): 26-32.
20. Stagnara P, De Mauroy JC, Dran G, et al. Reciprocal angulation of vertebral bodies in a sagittal plane: approach to references for the evaluation of kyphosis and lordosis. *Spine* 1982; **7**: 335-42.
21. Cowell HR, Hall JN, MacEwen GD. Genetic aspects of idiopathic scoliosis. *Clin Orthop* 1972; **86**: 121-31.

References continued at foot of next column

Medical Ethics

THE PHYSICIAN'S RESPONSIBILITY TO THE PATIENT*

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AS a university teacher of community medicine and health I do not have responsibility to individual patients. One of my duties is to protect people against the harm that over-enthusiastic doctors and misguided politicians can inflict upon them. My comments on the physician's responsibility to the patients are those of an outsider on the inside. I shall confine myself to two themes: the interference by the State in the doctor's care for his patients; and the ethical asymmetry between the doctor-initiated and the patient-initiated consultation.

MEDICINE AND THE STATE

Ideally, the doctor-patient relationship should be characterised by confidentiality, trust, truthfulness, honesty, loyalty, and beneficence. Sir Theodore Fox, a former editor of *The Lancet*, argued that "the physician is not the servant of science, or of the race, or even of life. He is the individual servant of his individual patients, basing his decisions always on their individual interest".¹ Unfortunately, these lofty aspirations do not exist in a vacuum. There is a third party, the State, which sees both the doctor and the patient as its servants.

The State can interfere directly with the doctor's responsibility to his patient by law: it can rule that euthanasia is illegal, that abortion is illegal, that prescription of addictive substances is illegal, thus forcing the doctor to choose between an action that may be in the best interests of his patient, albeit criminal, and the betrayal of his patient by accepting the role of an agent of the State. The State can muzzle the doctor by threats and sanctions, as happened to a senior medical officer in Bath who was suspended for criticising Government policy, or to a director of community medicine in Bedfordshire who was suspended for talking to the Press and leaking a confidential paper on AIDS prevention.² Dr Ann Dally was prevented, by the General Medical Council, from providing humane care for patients addicted to drugs, under the threat of having her medical licence revoked. The General Medical Council, described by one critic as prosecutor, judge, jury, and

executioner, is in some sense an extension of the arm of the State.³

According to the Declaration of Geneva of 1948, the doctor must respect the secrets that are confided to him by patients. The word "respect", however, does not mean that his lips are sealed. On the contrary, according to the Medical Defence Union, the doctor in Britain is obliged by law to disclose certain information confided to him, in the hope of secrecy, by a trusting patient. The legal requirement extends to nine specified situations. In one of them, under the Misuse of Drugs (Notification of, and Supply to Addicts) Regulations 1973, any doctor attending a patient whom he considers to be, or has reasonable grounds to suspect is, addicted to controlled drugs, must notify the Chief Medical Officer or the Home Office within seven days. That this order, making police informers out of doctors, has nothing to do with crime prevention can be seen from the fact that "if a doctor knows that a patient is about to commit, or has already committed, a crime, there is no general legal duty to inform the police".⁴

Less direct, but more damaging in its encompassing pervasiveness is the State's power to disseminate propaganda under the general heading of "health promotion and prevention". The doctor's responsibility to his patient includes protection of the patient from distortion of the concept of health, medicalisation of life, and false promises of disease prevention. Barsky⁵ showed how the increasing obsession with health in the USA has detracted from the sense of wellbeing in the population.

The medicalisation of health, a feature of totalitarian societies, serves as a justification for State intrusion into people's private lives, and for stigmatising those who do not conform. Sir Raymond Hoffenberg, President of the Royal College of Physicians, recently suggested that smokers and drinkers should pay for treatment and thus suffer a penalty.⁶ Other undesirable elements of society may be forced to undergo compulsory tests for diseases which, to use the words of Edwina Currie, "good Christians do not catch", or undergo compulsory "treatment", all in the name of improving "national health" (a term popular in the Germany of the 1930s). A "national diet" is seriously contemplated, and the State sponsors projects in "behavioural modification". It was Thomas Jefferson, in *The Notes on the State of Virginia* (1781), who wryly observed that "were the government to prescribe medicine and diet, our bodies would be in such keeping as our souls are now". We have made great advances since. The direction is clear to achieve the platonic ideal of society. I quote from Plato's *Republic*: "These are the kind of doctors and judges for whom you will legislate in your State. They will treat those of your citizens whose physical and psychological constitution is good; as for the others, they will leave the unhealthy to die, and those whose psychological constitution is incurably warped they will put to death."

*Based on a paper given at a workshop on Cost versus Benefit in Non-surgical Cancer Treatment, Camberley, April 15, 1988.

R. A. DICKSON: REFERENCES—continued

22. Tanner JM. Growth at adolescence. 2nd ed. Oxford: Blackwell, 1962.
23. Willner S. Spinal pantograph—a noninvasive anthropometric device for describing postures and asymmetries of the trunk. *J Pediatr Orthop* 1983; **3**: 245–49.
24. Dickson RA, Lawton JO, Butt WP. The pathogenesis of idiopathic scoliosis. In: Dickson RA, Bradford DS, eds. Management of spinal deformities. London: Butterworths International Medical Reviews 1984: 1–37.
25. Pool RD, Smith RM, Sharples D, Dickson RA. Bone drift and structural scoliosis. *Orthop Trans* 1987; **11**: 109–10.
26. Dickson RA, Archer IA. The biomechanics of spinal deformity. *J Bone Joint Surg* 1986; **68B**: 682.
27. Archer IA, Dickson RA. Stature and idiopathic scoliosis. *J Bone Joint Surg* 1985; **67B**: 185–88.
28. Mehta MH. Infantile idiopathic scoliosis. In: Dickson RA, Bradford DS, eds. Orthopaedics 2. Management of spinal deformities. London: Butterworths 1984: 101–20.
29. Lloyd Roberts GC, Pincott JR, McMeniman P, Bayley IJL, Kendall B. Progression in idiopathic scoliosis: a preliminary report of a possible mechanism. *J Bone Joint Surg* 1978; **60B**: 451–60.
30. Saartok T, Dahlberg E, Bylund P, Eriksson E, Gustafsson J. Steroid hormone receptors, protein and DNA in erector spinae muscle from scoliotic patients. *Clin Orthop* 1984; **183**: 197–207.
31. Zetterberg C, Aniansson A, Grimby G. Morphology of the paravertebral muscles in adolescent idiopathic scoliosis. *Spine* 1983; **8**: 457–62.
32. Smith RM, Dickson RA. Experimental structural scoliosis. *J Bone Joint Surg* 1987; **69B**: 576–81.

The Savage from Huxley's *Brave New World* died in despair. He wanted God, he wanted poetry, he wanted real danger, he wanted freedom, he wanted goodness, he wanted sin. When the Architect of Happiness asked incredulously, "Are you claiming the right to be unhappy, the right to have syphilis and cancer?" the Savage replied, "Yes, I claim them all". Sir Theodore Fox put it in different words, but not less effectively: "Life is not really the most important thing in life. Some cling to it as a miser to his money, and to as little purpose. Other wear it lightly—ready to risk it for a cause, a hope, a song, the wind on their face".¹

The doctor's responsibility to his patient is made even more difficult by the fact that doctors themselves are not immune to mental pollution by the health speak of the life-stylists. The gospel of salvation through health (by the year 2000) heard from the pulpits of politicians and their medical cronies serves as a red herring to distract attention from the real issues—inequality in health care; victim-blaming; poverty. Bertrand Russell noted, in his essay on vicarious asceticism, that "any section of mankind which has a monopoly of power is sure to invent theories to prove that the rest of mankind had better do without the good things in life". A State which preaches that, if only people would stop smoking, drinking, fornicating, and enjoying their food, cancer and heart disease would disappear, should heed the warning of the mad poet Hölderlin: "What has made the State into hell is that man wanted to make it its heaven". Cancer prevention and cancer screening, with its rhetoric of life-saving, is the latest addition to the false promises.

ETHICS OF SCREENING

This brings me to my second theme. When a person knocks at the door of the surgery, asking for help, that knock transforms him into a patient. His complaint becomes medicalised, and the doctor, without promising anything, consoles, cares, and sometimes cures. As opposed to this, when a doctor asks a healthy person to come in, he promises a benefit, he medicalises health, he solicits business. As Archie Cochrane once put it, he is like an evangelist calling "Come unto me, ye faithful, and I shall cure your ills". Promises entail obligations. The ethics of cancer screening are different from the ethics of an ordinary medical consultation.

In the patient-initiated encounter, the doctor, before embarking on invasive investigations or treatments, protects himself by asking the patient to sign a piece of paper testifying informed consent. In doctor-initiated screening, the patient should be similarly protected, by a written guarantee of benefit. If the doctor cannot in honesty sign it then he should not offer such a service. A failure to meet the terms of the signed guarantee would make the doctor responsible for a breach of contract.

There is no end to cancers for which any healthy person could be screened, and the shorter the intervals between screenings the better. This may be profitable to screeners but, in the absence of evidence that screening is beneficial, harmful to healthy people.

The Declaration of Helsinki, updated in Tokyo in 1975, on experimentation on human subjects, makes it clear that before any research project is undertaken, possible risks must be carefully evaluated. Furthermore, each subject must be fully informed about the nature and magnitude of these risks. If this has been done in major screening programmes, the fact is not mentioned in the published reports. Yet there is no doubt that cancer screening is an

experimental procedure of uncertain benefit: how else could randomised controlled trials of cancer screening be ethically justified. The hazards of screening are also undisputed: they include false-positive results leading to unnecessary investigations and treatments, with resulting iatrogenic morbidity, both physical and psychological, in people who were healthy. How many healthy individuals may be sacrificed for the putative collective benefit? The harms of screening are passed over in silence: to mention them could be counterproductive—people might not come next time. For one woman who may potentially benefit from cervical screening, 40 000 smears have to be taken and 100 women must be treated unnecessarily. In breast cancer screening, one woman out of 10 000 women screened per year may benefit. These are secrets which must be kept hidden from the women themselves—for obvious reasons.⁸

Not only are the ethics of screening quite different from the ethics of an ordinary medical consultation, governed by the old but not outdated precept of "first do no harm", but they are not even formulated. One of the reasons for this unhappy oversight is that the advocates of screening are often not medically qualified and may find it difficult to distinguish between statistical significance and clinical relevance. They present screening as an unmitigated good. Practising doctors accept these claims at face value and thus become unwitting accomplices in the rigged gamble.

Those two astute observers, Walter Holland and David Sackett, in their important article on screening, pointed out that "the advocates of screening, usually for impeccable motives, conclude that the pre-existing evidence plus commonsense, in face of the ongoing toll of disability and untimely death, demands massive screening programmes . . . In 'keeping the faith', screening advocates may find themselves forced to accept or reject evidence not so much on the basis of its scientific merit as on the extent to which it supports or rejects the stand that screening is good".⁹

The situation can be improved by providing ethical guidelines for screening. The Helsinki-Tokyo Declaration can be easily adapted to the purpose. Similar guidelines on ethics of human experimentation have been issued by the Royal College of Physicians in 1984.¹⁰ If individual human "guinea pigs" must be protected from excessive research zeal by having to be informed about the inherent risks, the same imperative should apply with even greater force when whole populations of healthy people are put at risk by screening. Moreover, as doctors are obliged by their ethical code to evaluate carefully the balance of harm and benefit of any procedure they offer to patients, the application of this rule would go a long way towards protecting their patients against the vested interests of screeners. Once this rule is adhered to, the screening bubble will burst.

CONCLUSION

The ideology behind the current health promotion rhetoric is an unhealthy mix of utopian and totalitarian thinking. In processing masses of healthy people through screening mills, under a false promise of cancer prevention, the doctor ceases to be the patient's advocate and becomes an agent of a State policy. His responsibility to individual patients is abrogated and substituted by the dictat of self-appointed health manipulators. The writing is on the wall.

THE EXPERIMENTAL BASIS OF IDIOPATHIC SCOLIOSIS.

Lawton JO, Dickson RA.

Clinical Orthopaedics; 210: 9-17, 1986.

A model of structural scoliosis in the thoracic region resembling that found clinically in patients was established in the rabbit by the production of an asymmetric thoracic lordosis. This buckled to the side with growth to cause a progressive deformity akin to the human situation. If the thoracic kyphosis was restored during growth then there was a progressive reduction in curve magnitude. This was the first reliable animal model of structural scoliosis and tested the hypothesis in the animal that restoration of the thoracic kyphosis was a physiological approach to treatment.

The Experimental Basis of Idiopathic Scoliosis

J. O. LAWTON, M.A., F.R.C.S.,* AND ROBERT A. DICKSON, M.A., CH.M., F.R.C.S.**

Idiopathic scoliosis is a complex three-dimensional deformity of the spine of which the scoliotic deformity and rotation are entirely secondary to an abnormality of shape in the median sagittal plane. In the thoracic region, there is a lordosis where a kyphosis should exist. In a controlled series of animal experiments using the growing New Zealand white rabbit as the model, idiopathic scoliosis can readily be produced if an asymmetric lordosis is created in the lower thoracic spine. Neither pure lordosis nor pure scoliosis produces the progressive idiopathic deformity. In the experimental animal when the normal thoracic kyphosis is reconstituted before maturity, the idiopathic deformity spontaneously improves. This lays the foundation for a more physiologic approach to treatment.

Idiopathic scoliosis is a three-dimensional deformity of the spine combining lateral curvature with vertebral body rotation. The essential lesion, however, lies in the median sagittal plane in the nature of a lordosis.^{1,26} Heuer¹⁶ extended this idea and considered that the lordosis resulted from overgrowth of the anterior vertebral column relative to the posterior elements. Somerville³⁸ renewed the concept, and Roaf,³⁴ describing the anatomy of the scoliotic deformity, emphasized that a lordotic region lies at the apex of the curvature. Supportive work is provided by Deane and Duthie⁹ and Perdriolle and Vidal.^{29,30} This

concept has been developed and clarified following epidemiologic, clinical, cadaveric, and biomechanical analyses^{11,12} that confirmed the presence of an apical lordosis and its rotational potential but indicated that asymmetry in the transverse or coronal plane was required to impart rotational instability to the lordosis.

Workers have tried to create a scoliotic deformity in the experimental animal, but the rationale behind the techniques has little in common with the situation in clinical idiopathic scoliosis. Division of muscles, nerves, ligaments, and bone produces a traumatic or paralytic type of curvature with little or no rotation,^{19,21,37} and premature fusion of vertebral growth zones has met with limited success.^{4,14,27} The presence of a lordotic region in the thoracic spine appears to be fundamental to the pathogenesis of idiopathic scoliosis, and thus it is important to establish this mechanism in the experimental animal.

The object of this study was to test the hypothesis in the experimental animal that asymmetric lordosis is the basic lesion of idiopathic scoliosis. This study was extended to investigate the principle that recreation of the thoracic kyphosis without fusion in an animal with a progressive scoliotic deformity would favor resolution of the scoliotic deformity and thus represent a more physiologic approach to treatment.

MATERIAL AND METHODS

Forty freshly weaned male New Zealand white rabbits, weighing 1.5 to 2 kg, were used. They were housed in individual cages and allowed to take water and a proprietary pellet diet *ad libitum*. Operations were performed under intravenous pentobarbitone

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TABLE 1. Experimental Animal Groups

<i>Spinal Asymmetry</i>	<i>No. of Animals</i>	<i>Method</i>	<i>Follow-up Results</i>
Median plane	10	Lordosis	Observed
Coronal plane	10	Lateral release	Observed
Biplanar	20	Lordosis plus Lateral release	10—observed 10—release lordosis

anesthesia augmented by local 1% xylocaine infiltration. Through a midline dorsal incision, a subperiosteal exposure of the spinous processes and laminae was performed bilaterally from the fifth to the tenth thoracic level. Median (sagittal) plane spinal asymmetry alone, imitating lordosis, was created in ten animals by approximation of the spinous processes using a braided nylon suture. Coronal plane asymmetry alone was created in ten animals by a lateral-releasing procedure, removing the ligamenta flava and facet joint capsules on the left side using local thermal coagulation. Combined median and coronal plane spinal asymmetry (an asymmetric lordosis) was created in the other 20 animals and was allowed to develop in ten of these 20 animals without intervention. In the other ten, the lordotic region was released when the developing scoliosis had progressed beyond a Cobb angle of 30°. The designation of the animal groups is summarized in Table 1.

The development of a spinal curvature was assessed on routine anteroposterior and lateral radiographs of the spine with the animal handheld against the cassette with the lower limbs extended. Radiographs were taken before operation and one, two, and three weeks after operation and every three weeks thereafter. Curve magnitudes were measured according to Whittle and Evans⁴¹ adaptation of the method of Cobb, and the degree of lordosis was determined by measurements across the endplates of the apical three vertebrae. The animals were

killed 15 weeks after the initial procedure with an intravenous overdose of pentobarbitone. The whole spine and thoracic cage was dissected free, removed, and examined macroscopically. A representative sample of rabbit spines was subjected to computed axial tomography.

RESULTS

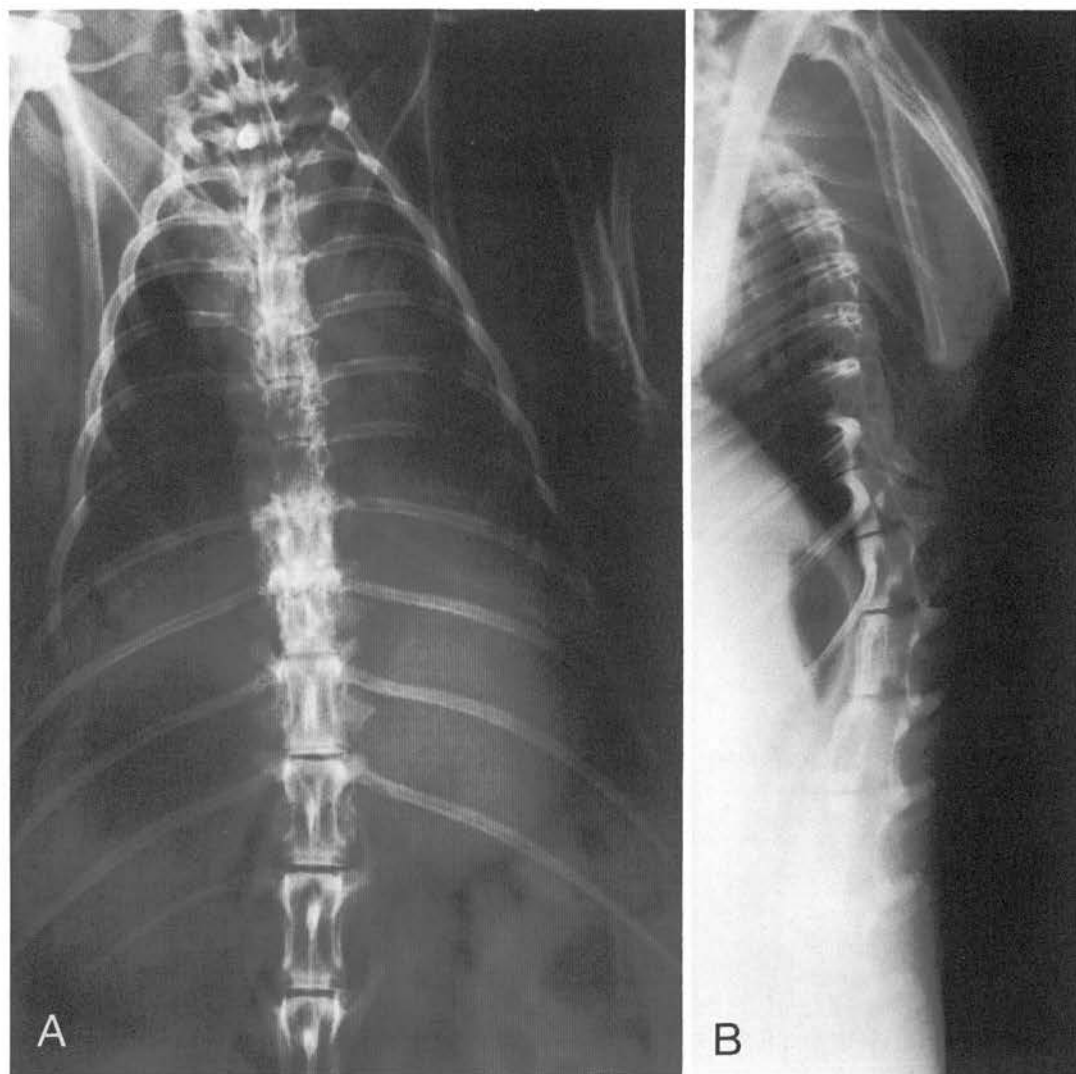
Three animals died shortly after surgery, two from the coronal plane asymmetry alone group and one from the biplanar asymmetry group, following release of the lordosis-retaining suture. Lateral radiographs demonstrated a loss of the initial lordosis following fracture of the spinous processes in four animals. This affected two animals from the median plane asymmetry alone group at three and six weeks after operation and two from the biplanar asymmetry group at one week and three weeks after operation.

Table 2 summarizes the initial outcome of the animals following operation. In all animals with median plane asymmetry alone, the spine remained straight in the coronal plane (Fig. 1). Radiographic and macroscopic examination confirmed the persistence of the lordosis in eight of the animals; in the other two, flattening of the normal thoracic curvature in this region existed despite postoperative loss of the lordotic segment.

Of the eight animals available for assessment from the group showing coronal plane asymmetry alone, only one developed a progressive scoliosis (74° at 12 weeks). In another three, a mild initial curvature occurred but did not progress to the same extent (mean, 35° at 12 weeks). All 20 animals in the combined median and coronal plane spinal asymmetry group developed a scoliotic deformity. Of the ten animals whose curvature was allowed to develop, eight progressed to a severe deformity with growth (Fig. 2). In one animal in which the lordosis was lost at three weeks (at a curve magnitude of 38°), the deformity stopped progressing, and in the animal in which lordosis was lost at one week, the scoliosis resolved. A summary of the overall development of the curvatures in these ten animals com-

TABLE 2. Scolioses Produced

<i>Spinal Asymmetry</i>	<i>Assessable</i>	<i>Outcome</i>	
		<i>Straight</i>	<i>Scoliosis</i>
Median plane	10	10	—
Coronal plane	8	4	4
Biplanar	20	—	20

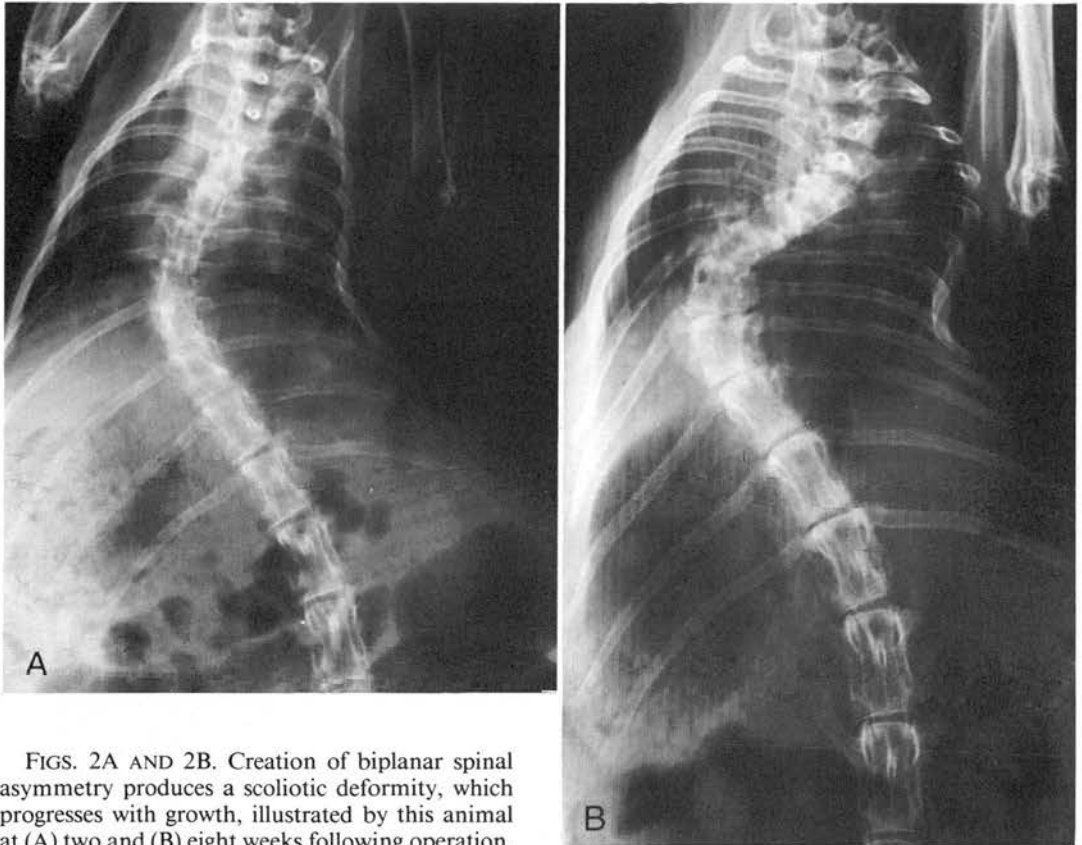


FIGS. 1A AND 1B. This animal from the median plane asymmetry group has maintained the lordotic region 12 weeks following operation but has failed to develop a scoliotic deformity.

pared with those with coronal plane asymmetry alone is shown in Table 3.

The lordosis-retaining suture was removed in ten animals with initial biplanar spinal asymmetry at one to two weeks following the first operation. Of the nine survivors, five animals showed regression of their scolioses (greater than 10° change in curve magnitude) with a mean curve improvement of 18° (Fig. 3). In three animals, the curvature continued

to progress (mean curve increase, 16°), while in one animal the deformity remained static. Lateral radiographs following lordosis release showed that all animals regained a kyphosis of the apex, although in only one animal was the preoperative extent of the kyphosis achieved. Mean scoliosis magnitude at the time of lordosis release was 18° greater in those that progressed than in those that regressed (42° and 60° , respectively). When the apical



FIGS. 2A AND 2B. Creation of biplanar spinal asymmetry produces a scoliotic deformity, which progresses with growth, illustrated by this animal at (A) two and (B) eight weeks following operation.

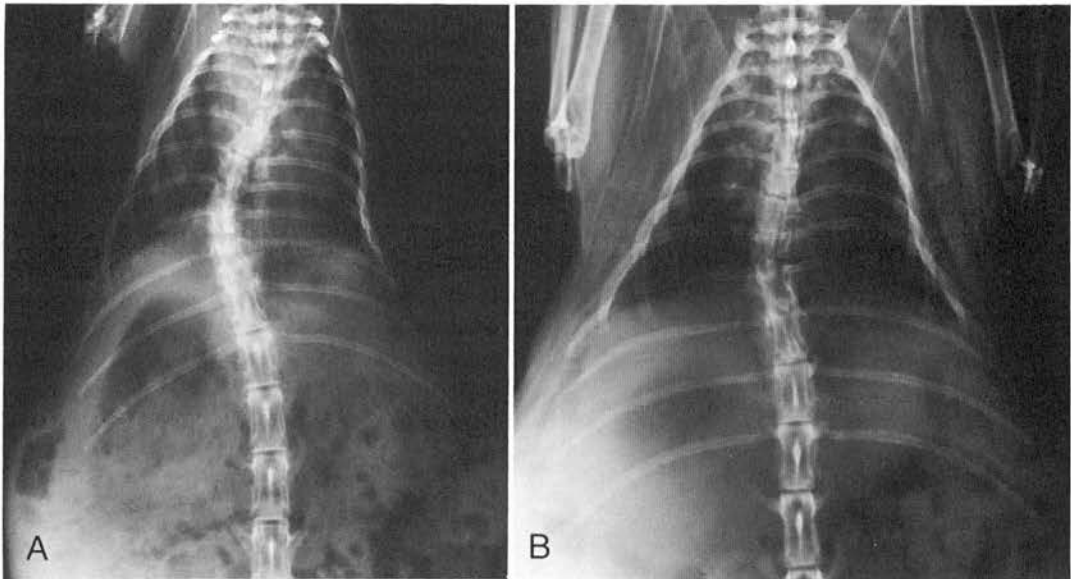
thoracic kyphosis following lordosis release was compared with the preoperative value, the mean difference was less in those animals whose curve regressed than in those whose curve progressed (3.6° and 8° , respectively). Table 4 summarizes the results in this group.

Macroscopic examination of the animals that developed a progressive scoliosis con-

firmed the lordoscoliotic nature of the thoracic curvature with vertebral rotation and a secondary rib hump (Fig. 4). Five of the specimens were subjected to computed axial tomography, which demonstrated a mean apical vertebral rotation of 19° (Fig. 5). Maceration of the specimens allowed a direct comparison of the ribs from the convex and concave sides.

TABLE 3. Magnitude of Scolioses

Spinal Asymmetry	No. Assessable	Mean Cobb Angle (+ Range) ($^{\circ}$) (Weeks After Operation)					
		0	1	3	6	9	12
Coronal plane	8	2 (0-12)	11 (0-32)	16 (0-52)	21 (0-67)	22 (0-69)	23 (0-74)
Biplanar	10	15 (0-32)	42 (15-64)	53 (13-74)	55 (0-80)	57 (0-87)	61 (0-108)



FIGS. 3A AND 3B. Following release of the lordosis-retaining suture, this rabbit demonstrates regression of the curvature with growth.

In addition to a difference in the overall curvature of the ribs, there was a demonstrable torsional deformity in each.

DISCUSSION

A wide variety of procedures has been used to try to create scoliosis in the experimental animal, the majority falling into two broad categories. In the first, stability of the spinal column has been affected by division of muscle, ligament, nerve, and bone. In the second, selective injury to vertebral growth plates has

been used to try to produce asymmetric growth of the spine.

Selective defunctioning of paraspinal musculature was carried out by Schwartzmann and Miles,³⁷ producing a paralytic curve convex to the operated side. Bisgard⁵ divided intercostal nerves in rabbits and goats and again produced a paralytic curve, although he found that this tended to correct with time. In a more comprehensive study by Liszka,²¹ selective division of both anterior and posterior, or simply posterior, spinal nerve roots in rabbits produced an immediate functional curve. He inferred

TABLE 4. Magnitude of Curves Following Lordosis Release

Outcome	No.	Curve Alteration (°)		Curve magnitude at Release (°)		Restoration* Apical Kyphosis (°)	
		Mean	Range	Mean	Range	Mean	Range
Regress	5	18	11-25	42	36-49	3.6	0-7
Static	1	—		39		5	
Progress	3	16	11-26	60	58-64	8	7-9

* Preoperative-postlordosis-release apical kyphosis.

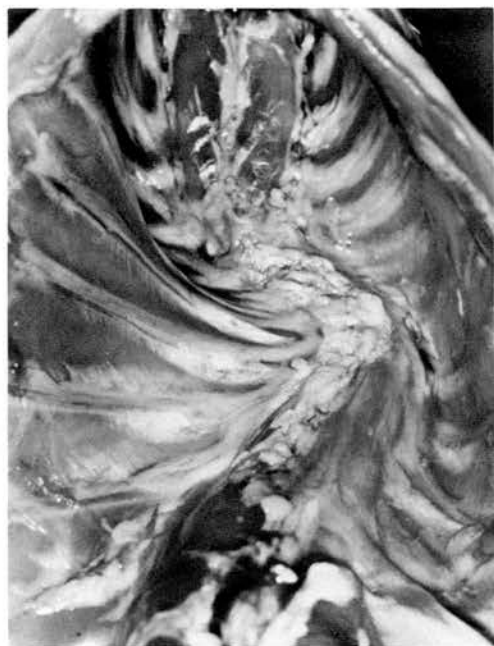


FIG. 4. A view taken looking into the thoracic cage from below illustrates the lateral curvature and vertebral body rotation in this specimen taken from an animal with a progressive scoliosis.

that division of the sensory input was sufficient to upset spinal reflexes and cause the lateral curvature. This work was repeated by MacEwen.²² However, when Alexander *et al.*² performed a similar study and subsequently examined the spinal cords microscopically, they found that a curvature was produced only where there was evidence of anterior horn chromatolysis. They concluded that the final common pathway remained interruption of the motor innervation.

A comprehensive study was carried out in rabbits and pigs by Langenskiöld and Michelsson,^{19,20} variously dividing muscles, ligaments, and ribs in an attempt to produce a scoliotic deformity. The most consistent results followed excision of the head and neck of the ribs, creating an immediate functional curve convex to the side of operation. They thought that the significant factor in the pathogenesis of these curves was division of the posterior costotransverse ligament, al-

though they admitted that a severe progressive curvature developed only in a few animals where this ligament was sectioned alone. Manning²⁵ and MacEwen^{22,23} repeated the work and found that division of this ligament alone had limited success. In a similar study using rabbits, Piggott^{31,32} concluded that failure of the development of the rib head was the significant factor in allowing a deformity to develop. However, the resection of rib heads in primates by Robin and Stein³⁵ failed to produce any significant deformity of the spine.

The established scoliotic spine exhibits both lateral deviation, with wedging of the vertebrae in the coronal plane, and vertebral body rotation and associated pedicle length asymmetry. Coronal plane wedging has been established by unilateral damage to the vertebral body growth plates but produces a minor lateral curvature with little or no rotation.^{3,6,14,27} Asymmetric pedicle growth secondary to an abnormality of the neurocentral junction was considered by Knutsson¹⁷ as a cause of vertebral rotation. Accordingly, when Ottander²⁸ fused the neurocentral junction unilaterally in the second lumbar vertebra of just one pig, he produced a minor scoliotic deformity with a few degrees of rotation. A similar approach by Beguiristain *et al.*,⁴ however, produced a significant deformity in only one out of eight pigs.

Other procedures used to create a scoliotic deformity in the experimental animal include creating a traumatic pleurisy,⁵ plaster-cast immobilization,^{15,18} feeding animals lathyrictic or rachitic diets,^{33,42} and producing labyrinthine dysfunction.^{24,43} It is difficult to implicate any of these mechanisms in the pathogenesis of clinical idiopathic scoliosis.

Following his clinical observations, Somerville³⁸ attempted to produce a scoliotic deformity in the experimental animal by creating a lordosis of the midthoracic spine; this was successful in three rabbits. In contrast, Wittebol⁴⁰ and Gottlieb¹³ had disappointing results. To impart rotational instability to a thoracic lordosis, asymmetry is required in another plane. Whereas spinal asymmetry in both the coronal and transverse planes exists

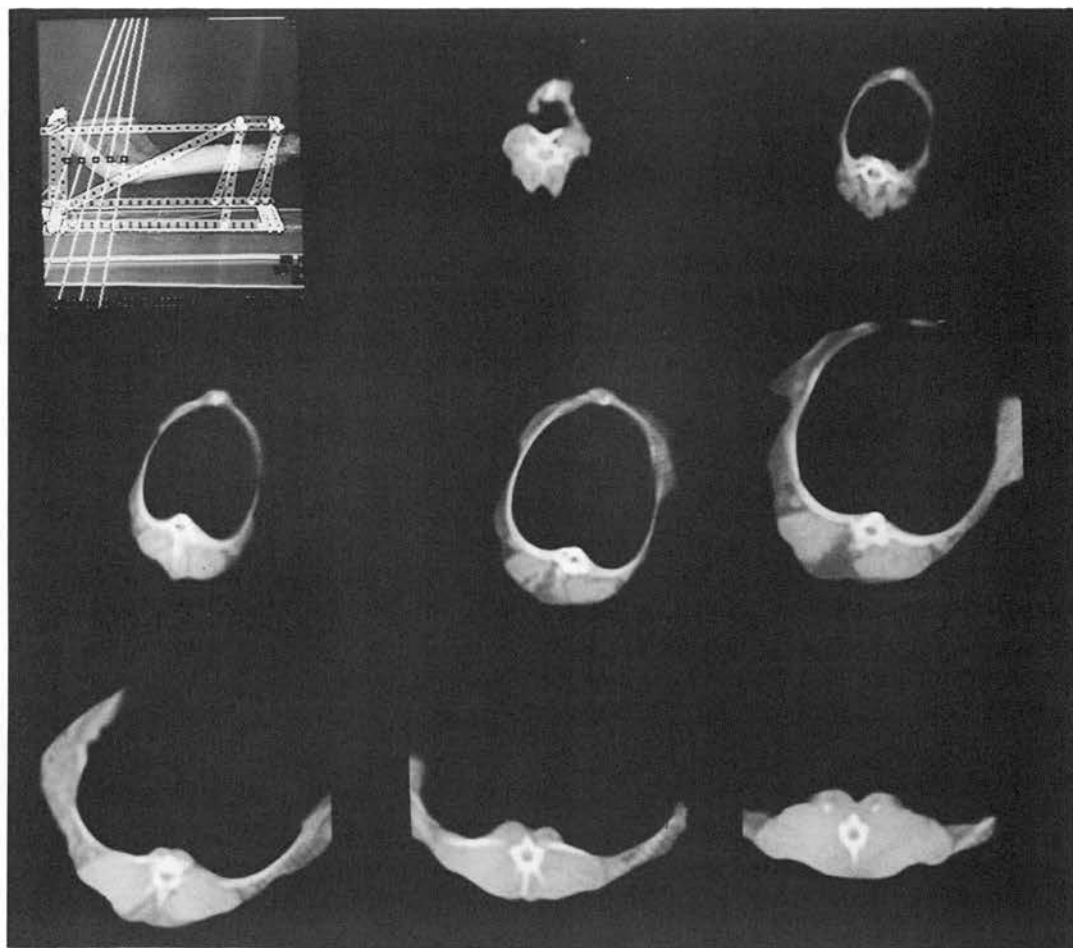


FIG. 5. Computed axial tomography of a postmortem specimen with scans taken from the cervicothoracic to thoracolumbar region confirms the convex side vertebral body rotation and associated rib hump.

in humans, this has not been recorded in experimental animals.^{7,8,36,39}

The results of this study confirm the importance of biplanar spinal asymmetry in producing a progressive scoliotic deformity. In the ten animals with median plane asymmetry alone, the spine remained straight during growth. Of the animals with coronal plane asymmetry alone, only one developed a progressive curve. It is probable that in this animal soft tissue fibrosis from the initial exposure was sufficient to create a relatively stiff region posteriorly, thus producing additional asym-

metry in the median plane. However, biplanar spinal asymmetry produced a scoliotic deformity in all 20 animals. The initial magnitude of the curve and its subsequent rate of progression confirms the mechanical importance of an asymmetrically lordotic segment.

Release of the lordotic segment occurred inadvertently in two animals, and its effect was significant. In the animal whose curve had already reached 38° by this time, the curve failed to progress. However, in the animal whose median plane spinal asymmetry was lost at an earlier stage, the scoliotic curvature resolved

with growth in the manner of the resolving infantile idiopathic case. When this lordosis-release procedure was performed deliberately, five of the nine animals available for assessment showed curve regression. It is interesting that the mean curve magnitude at time of lordosis release was considerably greater in the three animals whose curve progressed than in the five whose curve regressed; in the animal whose curvature remained static, the Cobb angle at release was intermediate. This suggests that beyond a certain curve magnitude, the deformity becomes autonomous. The latter supposition is supported by the radiographic analysis of the thoracic contour before operation and after lordotic release, where a more complete restitution of the thoracic kyphosis was achieved in those animals whose curves regressed.

This experiment illustrates a mechanical model in an animal that has already passed peak growth velocity (growth velocity is waning by the time weaning is over). The vertebral abnormalities akin to the reverse of Scheuermann's disease observed at the apex of early adolescent scoliotic deformities^{11,12} are presumably the result of minor repetitive growth disturbances. These would have a maximum influence on the development of the spinal deformity during the adolescent growth spurt, during which phase idiopathic scoliosis is most prevalent.¹⁰ Such a favorable situation was not achievable in this model, in which the "growth disturbance" had to be achieved at one sitting and at a time when peak growth velocity is waning. With the maximum influence of growth already past, it followed that the major part of any progressive deformity had developed within the first few weeks after operation; similarly, it was not surprising in the lordosis-release group that following initial curve improvement a deformity remained. An additional factor tending to inhibit the development of a scoliotic deformity is the dependent thorax of the quadruped, that tends to resist vertebral rotation. However, the creation of an unstable spinal configuration secondary

to combined median and coronal plane spinal asymmetry has been demonstrated.

This study confirms that biplanar spinal asymmetry is required to produce a progressive "idiopathic-type" scoliotic deformity. Such asymmetry produces rotational instability of the spine, the lordotic segment rotating away from the median plane and producing a scoliotic deformity as a secondary phenomenon. The spinning moment is not only produced by forward flexion of the thoracic spine but also develops during growth where the front of the vertebral body is gaining height faster than the posterior aspect. In the latter situation, to avoid developing a progressive lordosis, the excessive anterior vertebral body height can be accommodated only by the bodies buckling out to the side. This concept is of fundamental importance to the understanding of the pathogenesis of clinical idiopathic scoliosis and allows a more rational approach to treatment. Thus far, the operative management of idiopathic scoliosis concentrates on obtaining a partial correction of the secondary coronal plane deformity and protecting it from the deforming forces of subsequent growth by bony fusion. The observation from this study that early recreation of the thoracic kyphosis allows growth to be harnessed in favor of developing a straight spine lays the foundation for a more physiologic approach to the treatment of idiopathic scoliosis.

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REFERENCES

1. Adams, W.: Lectures on the Pathology and Treatment of Lateral and other Forms of Curvature of the Spine. London, Churchill & Sons, 1865.
2. Alexander, M. A., Bunch, W., and Ebberson, S. O. E.: Can experimental dorsal rhizotomy produce scoliosis? *J. Bone Joint Surg.* 54A:1509, 1972.
3. Arkin, A. M., and Simon, N.: Radiation scoliosis. An experimental study. *J. Bone Joint Surg.* 32A:396, 1950.
4. Beguiristain, J. L., DeSalis, J., Oriaifo, A., and Can-

- adell, J.: Experimental scoliosis by epiphysiodesis in pigs. *Int. Orthop.* 3:317, 1980.
5. Bisgard, J. D.: Experimental thoracogenic scoliosis. *J. Thoracic Surg.* 4:435, 1935.
6. Bisgard, J. D., and Musselman, M. M.: Scoliosis—Its experimental production and growth correction; growth and fusion of vertebral bodies. *Surg. Gynecol. Obstet.* 70:1029, 1940.
7. Burwell, R. G., James, N. J., Johnson, F., Webb, J. K., and Wilson, Y. G.: Standardised trunk asymmetry scores. A study of back contour in healthy schoolchildren. *J. Bone Joint Surg.* 65B:452, 1983.
8. Cruveilhier, J.: *Descriptive Anatomy*. London, Whitaker, 1841, p. 38.
9. Deane, G., and Duthie, R. B.: A new projectional look at articulated scoliotic spines. *Acta Orthop. Scand.* 44:351, 1973.
10. Dickson, R. A.: Scoliosis in the community. *Brit. Med. J.* 286:615, 1983.
11. Dickson, R. A., Lawton, J. O., Archer, I. A., Butt, W. P., Jobbins, B., Berkin, C. R., Bliss, P., and Somerville, E. W.: Combined median and coronal plane asymmetry: The essential lesion of progressive idiopathic scoliosis. *J. Bone Joint Surg.* 65B:368, 1983.
12. Dickson, R. A., Lawton, J. O., Archer, I. A., and Butt, W. P.: The pathogenesis of idiopathic scoliosis—Biplanar spinal asymmetry. *J. Bone Joint Surg.* 66B:8, 1984.
13. Gottlieb, O.: Experimental scoliosis (advance report). *Acta Orthop. Scand.* 26:295, 1957.
14. Haas, S. L.: Experimental production of scoliosis. *J. Bone Joint Surg.* 21:963, 1939.
15. Hakkarainen, S.: Experimental scoliosis: Production of structural scoliosis by immobilisation of young rabbits in a scoliotic position. *Acta Orthop. Scand.* (Suppl. 192): 1981.
16. Heuer, F.: *Zur Theorie der Skoliose*. Hessisches Artzeblatt 18:281, 1927.
17. Knutson, F.: A contribution to the discussion of the biological cause of idiopathic scoliosis. *Acta Orthop. Scand.* 33:98, 1963.
18. Langenskiöld, A.: Growth disturbance of muscle: A possible factor in the pathogenesis of scoliosis. In Zorab, P. A. (ed.): *Scoliosis and Growth*. Edinburgh, Churchill-Livingstone, 1971, pp. 85–90.
19. Langenskiöld, A., and Michelsson, J. E.: Experimental progressive scoliosis in the rabbit. *J. Bone Joint Surg.* 43B:116, 1961.
20. Langenskiöld, A., and Michelsson, J. E.: The pathogenesis of experimental progressive scoliosis. *Acta Orthop. Scand.* (Suppl 59): 1962.
21. Liszka, O.: Spinal cord mechanisms leading to scoliosis. *Acta Med. Pol.* 2:45, 1961.
22. MacEwen, G. D.: Experimental scoliosis. In Zorab, P. A. (ed.): *Proceedings of a Second Symposium on Scoliosis: Causation*. Edinburgh, E. & S. Livingstone, 1968, pp. 18–20.
23. MacEwen, G. D.: Experimental scoliosis and kyphosis. In Keim, H. A. (ed.): *Third Annual Postgraduate Course on Management and Care of the Scoliosis Patient*. New York, New York Orthopaedic Hospital, 1971, p. 76.
24. Magnus, R.: *Körperstellung*. Berlin, Springer, 1924.
25. Manning, C. W.: Experimental scoliosis. In Zorab, P. A. (ed.): *Proceedings of a Second Symposium on Scoliosis: Causation*. Edinburgh, E. & S. Livingstone, 1968, pp. 11–14.
26. Meyer, G. H.: *Die Mechanik der Skoliose*. Virchows Arch. [A.] 35:225, 1866.
27. Nachlas, I. W., and Borden, J. N.: Experimental scoliosis—The role of the epiphysis. *Surg. Gynecol. Obstet.* 90:672, 1950.
28. Ottander, H. G.: Experimental progressive scoliosis in a pig. *Acta Orthop. Scand.* 33:91, 1963.
29. Perdiolle, R.: *La scoliose: Son étude tridimensionnelle*. Paris, Maloine, 1979.
30. Perdiolle, R., and Vidal, J.: Étude de la courbure scoliotique. Importance de l'extension et de la rotation vertébrale. *Rev. Chir. Orthop.* 67:25, 1981.
31. Piggott, H.: Experimentally produced scoliosis in animals. In Zorab, P. A. (ed.): *Proceedings of a Second Symposium on Scoliosis—Causation*. Edinburgh, E. & S. Livingstone, 1968, pp. 15–17.
32. Piggott, H.: Posterior rib resection in scoliosis. A preliminary report. *J. Bone Joint Surg.* 53B:663, 1971.
33. Ponseti, I. V., and Shepard, R. S.: Lesions of the skeletal and other mesodermal tissues in rats fed sweet pea (*Lathyrus odoratus*) seeds. *J. Bone Joint Surg.* 36A:1031, 1954.
34. Roaf, R.: The basic anatomy of scoliosis. *J. Bone Joint Surg.* 48B:786, 1966.
35. Robin, G. C., and Stein, H.: Experimental scoliosis in primates—Failure of a technique. *J. Bone Joint Surg.* 57B:142, 1975.
36. Sabatier: *Traité complète d'anatomie*. Paris, 1777. (Cited by Farkas, A.: *Physiological scoliosis*. *J. Bone Joint Surg.* 23:607, 1941.)
37. Schwartzmann, J. R., and Miles, M.: Experimental production of scoliosis in rats and mice. *J. Bone Joint Surg.* 27:59, 1945.
38. Somerville, E. W.: Rotational lordosis: The development of the single curve. *J. Bone Joint Surg.* 34B: 421, 1952.
39. Taylor, J. R.: Scoliosis and growth. Patterns of asymmetry in normal vertebral growth. *Acta Orthop. Scand.* 54:596, 1983.
40. Wittebol, P.: Idiopathic scoliosis (an experimental investigation). *Arch. Chirurg. Neerland* 8:269, 1956.
41. Whittle, M. W., and Evans, M.: Instrument for measuring the Cobb angle in scoliosis. *Lancet* 1:414, 1979.
42. Yamamoto, H.: Experimental scoliosis in rachitic bipedal rats. *Tokushima J. Exp. Med.* 13:1, 1966.
43. Yamada, K., Yamamoto, H., Ikata, T., Nakagawa, Y., Kinoshita, I., Tezuka, A., and Tamura, T.: A neurological approach to the etiology and therapy of scoliosis. *J. Bone Joint Surg.* 53A:197, 1971.

Skeletal Metastases

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During the past ten to 20 years, there have been many advances in the understanding of the way in which skeletal metastases develop and great strides in the methods of detection, particularly with the development of the gamma camera and axial or whole-body skeletal scintigraphy. Skeletal metastases may present with pain, hypercalcemia, large lytic lesions, pathologic fracture, spinal cord or cauda equina compression, or spinal instability. Much has been learned about the management of skeletal metastases, and many of these developments have occurred in Great Britain.

DETECTION OF SKELETAL METASTASES

For many years it has been known that skeletal scintigraphy was more sensitive than standard radiographic techniques for the detection of skeletal metastases. The earlier studies utilized either a rectilinear scanner or external counting, and most relied on ^{47}Ca or ^{85}Sr as the radionuclide. The technique was very time-consuming, and only very limited areas of the skeleton could be examined. Skeletal scintigraphy, therefore, was limited to examining regions where skeletal metastases were suspected on clinical grounds. Galasko *et al.*^{31,53} reported a technique of axial skeletal scintigraphy using a gamma camera and ^{18}F . With this technique, the axial skeleton (skull, cervical spine, dorsal spine, lumbar spine, pelvis, rib cage, proximal humeri, and proximal femora) could be examined. They showed that

skeletal scintigraphy could be used not only for the detection of symptomatic skeletal metastases, but also for screening purposes. In 1969, Galasko described the results of axial skeletal scintigraphy in 50 women with advanced mammary carcinoma. Lesions were apparent on the radiographs in 25 (50%) and on the scintigram in 42 (84%). This incidence is similar to that reported by Jaffe,⁶³ who found skeletal metastases at autopsy in 85% of women who died from mammary carcinoma. Thirty-six (72%) of the 50 patients, including 17 who had no skeletal metastases evident on roentgenogram, had additional lesions present on the scintigram. Five patients were lost to follow-up examination; the scintigraphic findings were confirmed on subsequent roentgenograms or at autopsy in the other 31 patients.³² The time taken for the lesions to be confirmed radiographically varied from two to 18 months and at autopsy from one to nine months.

Since the late 1960s, there have been many reports confirming the accuracy and sensitivity of skeletal scintigraphy in the detection of skeletal metastases.⁴³ Edelstyn and colleagues²⁷ showed that at least 50% of the bone must be destroyed in the beam axis of the roentgen ray before a lesion involving the medulla can be seen radiographically. In contrast, lesions that involved the cortex were detected when very much smaller. Skeletal metastases develop in the medulla and cortical damage occurs late.

Clinical and biochemical parameters are also inaccurate. Galasko³² studied 86 patients with advanced mammary carcinoma, all of whom had skeletal metastases evident on roentgenogram. Only 65% of the patients complained of pain; when present, it was re-

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EXPERIMENTAL STRUCTURAL SCOLIOSIS.

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The rabbit model of structural scoliosis (Reference 21) was not subjected to study of the spinal cord and it was conceivable that a part of the operative procedure may have caused cord damage and thus a neuromuscular rather than an idiopathic-type scoliosis. This study therefore looked at the histopathology of the spinal cord and demonstrated that the various different mechanisms of production of experimental scoliosis put forward previously had the final common pathway of spinal cord damage. It was verified that the production of an asymmetric lordosis produced a scoliosis purely by mechanical and not neuromuscular means. Animals with the most progressive deformities failed to thrive, and developed cyanosis, being similar to the cardio-respiratory embarrassment of severe scoliosis in young children.

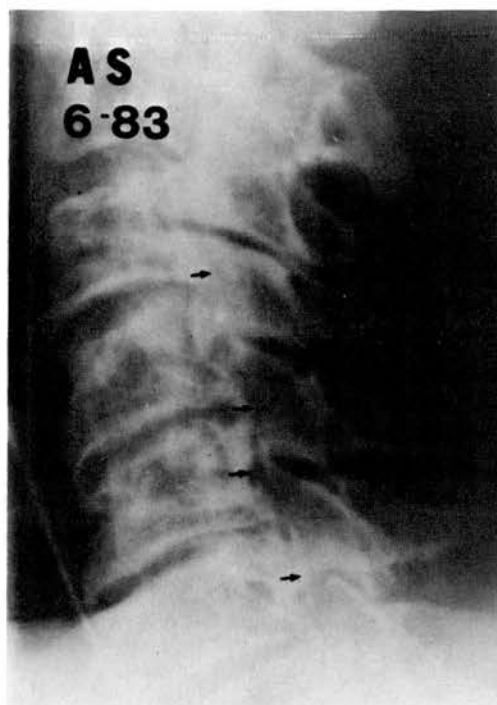


Fig. 5B

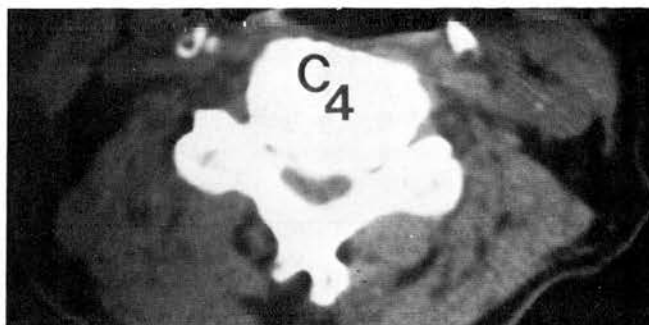


Fig. 5A

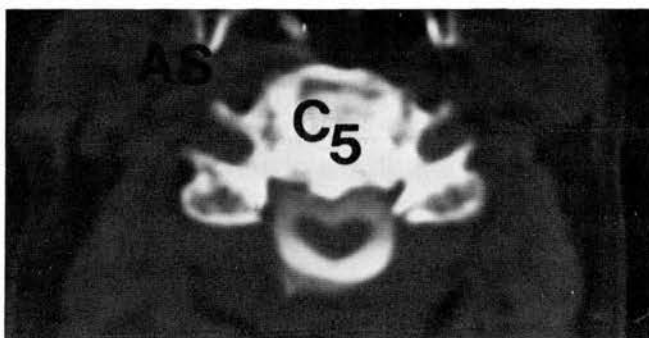


Fig. 5C

Case 5. This 72-year-old physician presented with a four-month history of a progressively unsteady gait. A – The lateral radiograph shows a segmental pattern of OPLL with radiopaque masses of bone extending into the spinal cord at C3, C4, C5 and C6. B – The pre-operative transaxial metrizamide CT image at the C4 vertebral level shows profound flattening of the spinal cord, with no contrast material between the OPLL and the anterior aspect of the spinal cord. C – The metrizamide CT image at the C5 level shows the appearance after wide decompression laminectomy from C2 to C7. The spinal cord and thecal sac have expanded posterior to the facet joints and contrast material flows anterior to the spinal cord. Two months after surgery the paraesthesia in his hands had resolved and his strength improved to Grade 5 in the right leg and Grade 4 in the left.

existing OPLL. Twelve of our patients in this series presented with cervical myelopathy and seven of the patients were tetraparetic and wheelchair-bound before a diagnosis of OPLL was made. The average time between the onset of tetraparesis and the diagnosis of OPLL was 17 months.

The value of NMR and multiplane CT scanning was demonstrated both in diagnosis and in pre-operative planning. To prevent delay in the diagnosis of severely tetraparetic and wheelchair-bound patients, the diagnosis of OPLL should be considered more often in patients who present with neurological symptoms which are not explained by other causes.

REFERENCES

- Bakay L, Cares HL, Smith RJ. Ossification in the region of the posterior longitudinal ligament as a cause of cervical myelopathy. *J Neurol Neurosurg Psychiatr* 1970;33:263–8.
- Becker DH, Conley FK, Anderson ME. Quadriplegia associated with narrow cervical canal ligamentous calcification and ankylosing hyperostosis. *Surg Neurol* 1979;11:17–9.
- Chin WS, Oon CL. Ossification of the posterior longitudinal ligament of the spine. *Br J Radiol* 1979;52:865–9.
- Forestier J, Rotes-Querol J. Senile ankylosing hyperostosis of the spine. *Ann Rheumat Dis* 1950;9:321–30.
- Goldwin RL. Calcified plaque in the cervical spine with pain and parasthesia. *JAMA* 1979;241:601–2.
- Hyman RA, Merten CW, Liebeskind AL, et al. Computed tomography in ossification of the posterior longitudinal spinal ligament. *Neuroradiology* 1977;13:227–8.
- Key CA. On paraplegia depending on disease of the ligaments of the spine. *Guy's Hospital Report* 1838;3:17–34.
- Murakami N, Muroga T, Sobue I. Cervical myelopathy due to ossification of the posterior longitudinal ligament: a clinicopathologic study. *Arch Neurol* 1978;35:33–36.
- Onji Y, Akiyama H, Shimomura Y, Ono K, Hukuda S, Mizuno S. Posterior paravertebral ossification causing cervical myelopathy: a report of eighteen cases. *J Bone Joint Surg [Am]* 1967;49-A:1314–28.
- Ono K, Ota H, Tada K, Hamada H, Takaoka K. Ossified posterior longitudinal ligament: a clinicopathologic study. *Spine* 1977;2:128–38.
- Resnick D, Guerra J, Robinson CA, Vint VC. Association of diffuse idiopathic skeletal hyperostosis (DISH) and calcification and ossification of the posterior longitudinal ligament. *AJR* 1978;131:1049–53.
- Rozario RA, Levine H, Stein BM. Cervical myelopathy and radiculopathy secondary to ossification of the posterior longitudinal ligament. *Surg Neurol* 1978;10:17–20.
- Tsukimoto H. A case report: autopsy of syndrome of compression of the spinal cord owing to ossification within the cervical spinal canal. *Arch Jap Chir* 1960;29:1003–7. (In Japanese.)
- Tsuyama N. Ossification of the posterior longitudinal ligament of the spine. *Clin Orthop* 1984;184:71–84.
- Tsuyama N, Terayama K, Ohtani K, et al. The ossification of the posterior longitudinal ligament of the spine (OPLL). *J Jpn Orthop Ass* 1981;55:425–40.

EXPERIMENTAL STRUCTURAL SCOLIOSIS

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Progressive structural scoliosis in growing rabbits has been produced. Tethering the thoracic spine into the form of an asymmetric lordosis produces a slowly progressive structural scoliosis by purely mechanical means. The addition of a contralateral release of the paraspinal muscles leads to a very progressive deformity with early cardiorespiratory failure. This release, however, was performed with an electric soldering iron and subsequent study showed that in those animals with severe progressive deformity there was localised spinal cord damage. We suggest that it is this neural damage and not the muscle release which leads to rapid progression. The clinical implications are important in that neurological dysfunction seems to render the spinal column less able to resist mechanical buckling and may be the crucial factor differentiating severely progressive from more benign curves.

Knowledge of the pathogenesis of structural scoliosis, particularly its idiopathic form, has been hampered by the lack of a consistent and explicable animal model. While the administration of mutagenic agents to pregnant animals readily produces a congenital spinal deformity (Duraiswami 1952; Ingalls and Curley 1957), and the postnatal induction of rickets (Yamamoto 1966) or lathyrism (Ponseti 1957) produces a metabolic form of spinal deformity by way of disordered collagen metabolism, such models tell us little about the development of the idiopathic deformity.

As in the clinical situation, most workers have addressed the more obvious lateral spinal curvature. The many procedures performed with the aim of resection or tethering of bone or soft tissue on one or other side of the spine (Bisgard 1934-5; Engel 1939; Haas 1939; Bisgard and Musselman 1940; Schwartzmann and Miles 1945; Miles 1947; Arkin and Simon 1950; Nachlas and Borden 1950; Bobechko 1973) usually fail to produce a spinal deformity at all or cause a minor non-progressive non-structural tilt of the spine in the coronal plane only.

Progressive structural scoliosis has only been produced in animals by two sorts of procedure. Resection of rib heads or adjacent ligaments, although both inconsistent and inexplicable in its effect, attracted much attention 20 years ago (Langenskiöld and Michelson 1961, 1962; Michelson 1965; Manning 1968;

Piggott 1968). The other method, the production of neurological damage at either cord or root level, has more consistently produced a paralytic type of scoliosis (Liszka 1961; Robin 1966; MacEwen 1968; Alexander, Bunch and Ebbesson 1972; Taffs, Magrath and Lytton 1979; De Salis, Beguiristain and Cañadell 1980; Pincott, Davies and Taffs 1984). While being excellent models of neuromuscular spinal deformities they tell us nothing about the production of the idiopathic form of scoliosis.

Structural scoliosis is, however, not a simple lateral curvature of the spine but a three-dimensional deformity with the lateral curvature component being the product of lordosis and rotation (Dick 1864; Adams 1865; Somerville 1952; Roaf 1966; Dickson et al. 1983, 1984). Somerville (1952) was the first to produce a progressive structural scoliosis in the growing animal by tethering the back of the spine into a lordosis but was successful in only three animals. He approximated the spinous processes by suture and added laminar cautery to reduce posterior spinal growth. We have followed his example in Leeds for the past six years and have used the same animal, the growing New Zealand White rabbit. Frustratingly, in 100 animals whose thoracic spine was tethered into a short-segment lordosis by approximation of either spinous processes or laminae on each side using wire sutures, the spines remained straight throughout growth (Lawton, Butt and Dickson 1983; Lawton, Scott and Dickson 1985). Wittebol (1956) and Gottlieb, Jørgensen and Movin (1957) had been just as disappointed when repeating Somerville's work. Only when the lordosis was made asymmetrical in the coronal plane by a lateral soft-tissue release with laminar cautery did it buckle to the side with growth to produce a three-dimensional deformity (Lawton et al. 1983). Although the lateral releasing procedure on its own did not produce a progressive deformity, it was nonetheless apparent that

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not inconsiderable biplanar spinal damage was necessary and that the lateral releasing procedure might have impinged upon the work of Langenskiöld and Michelson (1961, 1962) and those that followed.

Accordingly, this present study addresses three important points. Is there a less traumatic experimental animal model which reliably produces structural scoliosis by mechanical means only; is there a common pathological denominator which links the lateral releasing procedures of Langenskiöld and others with those who have produced neurological damage; and what is the clinical relevance of these animal experiments?

MATERIALS AND METHODS

Freshly weaned New Zealand White rabbits were used and received a standard pelleted diet and fresh water throughout the experimental period. All the operations were performed under intravenous pentobarbitone general anaesthesia supplemented with local infiltration of bupivacaine. Because previous experience (Lawton et al. 1983, 1985) had demonstrated that lordotic tethering of the spinous processes produced a symmetrical lordosis which failed to buckle with growth, a technique was developed in order to produce an asymmetric lordosis without the need for an additional lateral "releasing" procedure. Twenty-five animals underwent this new procedure. A further 20 animals underwent this new procedure and a lateral "releasing" procedure to render the lordosis even more asymmetrical in the coronal plane. In nine animals the lateral "releasing" procedure alone was performed (i.e. in the presence of a normal thoracic kyphosis).

Operative technique. The new procedure involved a posterior exposure of the thoracic spine and at the level of T4 and T10 the paraspinal muscles were gently retracted so that microfenestrations through the ligamentum flavum could be made above and below these vertebrae for the passage of the sublaminar suture. This was in the form of a 2/0 Ethibond suture (Ethicon W6937) which was passed under the lamina on one side of T4 and then over the paraspinal muscles down to the lamina of T10 on the same side, under which it was passed. The suture was then tied to draw the spine into a lordosis. The intervening section between T4 and T10 was undisturbed and care was taken not to overtighten the suture such that either the facet joints would be locked or the suture would bowstring to the side and act as a lateral rather than posterior tether. The side of the procedure (right or left) was varied at random. This procedure, performed in 25 animals (Group A), thereby produced a lordosis with directional instability.

In 20 further animals a lateral "releasing" procedure was added. This was performed by the application of local heat to the apical three laminae on the opposite side to the suture using an electric soldering iron until the bone was clean of muscle, the method used by Somerville

(1952) and one that we had used extensively previously to produce a lateral curvature of the spine (Lawton et al. 1985). These 20 animals formed Group B.

In a further nine animals (Group C) this lateral "releasing" procedure was performed alone (in the presence of a normal thoracic kyphosis).

The shape of the spine was assessed by weekly postero-anterior (PA) and lateral radiographs in a normal "relaxed" position. The degree of spinal curvature was measured using Cobb's method (Whittle and Evans 1979). The animals were sacrificed at five weeks by an overdose of intravenous pentobarbitone and the spine studied morphologically and histologically. Representative cross-sections of the spinal cord above, through and below the deformity were stained with Luxol fast blue counterstained with haematoxylin and eosin to demonstrate myelin in the spinal cord and thus establish cord integrity.

Table 1. The development of a deformity in Groups A, B and C. All measurements are the mean maximum measurable Cobb angle (in degrees), positive figures representing lordosis, negative figures kyphosis

Group	Pre-operative	Postoperative		
		One week	Three weeks	Five weeks
A. (progressors)				
Scoliosis	Nil	15.3	37.8	56
Lordosis/kyphosis	-17.4	28.5	36.8	36.6
A. (non-progressors)				
Scoliosis	Nil	Nil	Nil	Nil
Lordosis/kyphosis	-12.9	13.1	-10.8	-9.4
B. Scoliosis	Nil	57.2	87.3	107*
Lordosis/kyphosis	-16.5	Not measurable due to rotation		
C. (progressors)				
Scoliosis	Nil	73	103	110*
Lordosis/kyphosis	-15	Not measurable due to rotation		
C. (resolvers)				
Scoliosis	Nil	47	25	<3
Lordosis/kyphosis	-9.5	-26	-27	-2

* The gross deformity present by this stage made the Cobb angle measurement very inaccurate

These spinal deformities in the coronal and sagittal planes in the three groups A, B and C are shown in Table I. Four animals in Group A died and eight lost or failed to develop a lordosis. None of these animals developed a significant or progressive scoliosis. The remaining animals in Group A all developed a slowly progressive lordoscoliosis, always concave to the side of the suture and reaching a mean Cobb angle of 56° five weeks after surgery. Figures 1 to 4 show the normal pre-operative PA view of one of these Group A animals and the development of the deformity at one, three and five weeks. By five weeks there is a moderate scoliosis with rotation of the posterior elements into the curve concavity. The corresponding lateral views are shown in Figures 5 to 8. The normal thoracic kyphosis has been



Fig. 1



Fig. 2



Fig. 3



Fig. 4



Fig. 5



Fig. 6



Fig. 7



Fig. 8

Figures 1 and 5 – Normal.

Group A. Figures 2 to 4 – PA radiographs one, three and five weeks after surgery. Figures 6 to 8 – Lateral radiographs one, three and five weeks after surgery.

converted into a significant lordosis at one week which progresses with growth but after five weeks significant apical rotation has obscured the degree of lordosis on the lateral radiograph.

In Group B six of the animals died but 12 developed a rapidly progressive scoliosis, concave to the side of the suture, and thus convex to the side of the laminar cautery, reaching a mean Cobb angle of over 100° after five weeks. At this time the deformity was so great that simple Cobb angle measurement as perceived on the PA radiograph was no longer an accurate measure of the three-dimensional deformity. The remaining two animals in this group developed a slowly progressive deformity similar to the animals in Group A. Figures 9 to 14 show the development of the rapidly progressive deformity on PA and lateral radiographs one, three and five weeks after surgery.

Seven of these severely scoliotic animals were significantly growth retarded and four of these developed clinical cyanosis, the normal pink albino eye developing a dark blue colour. Arterial blood gas measurement

showed a severe respiratory failure with a pH of 7.34 (normal 7.45), $P_a\text{CO}_2$ of 8.52 mmHg (normal 4.56) and a $P_a\text{O}_2$ of 4.16 mmHg (normal 11.52). This was therefore a hypoxic, hypercapnic form of failure.

Two of the nine animals in Group C died; of the remaining seven, three developed a rapidly progressive scoliosis similar to the animals in Group B and four developed an initial scoliosis that resolved with growth. This resolution was always associated with persistence of the normal thoracic kyphosis.

Histological examination of transverse sections through the apex of the curve demonstrated the consequences of the laminar cautery. Although the intention of this had been to produce a lateral curvature of the spine by releasing the tension on the paraspinal muscles, all the animals in Group C, and the 12 in Group B with the severely progressive deformity, showed marked damage to the dorsolateral quadrant of the spinal cord (Fig. 15). In contradistinction, no evidence of cord damage was observed in the Group A animals and the two in Group B with the slowly progressive curve. In



Fig. 9



Fig. 10



Fig. 11

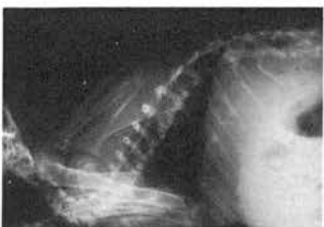


Fig. 12



Fig. 13



Fig. 14

Group B. Figures 9 to 11 – PA radiographs one, three and five weeks after surgery. Figures 12 to 14 – Lateral radiographs one, three and five weeks after surgery.

the animals where cord damage existed, it was directly related to the lateral cauterising procedure in terms of both site and side. Histomorphological examination at these sites of cord damage showed that this was the result of either direct thermal injury or cautery ablation of the

segmental vessel supplying the dorsolateral quadrant of the cord at that level. This vessel is intimately related to the lateral portion of the lamina, transverse process, rib head and costotransverse ligament.

DISCUSSION

In 1952 Somerville produced the first experimental animal evidence that the primary production of a lordosis led with growth to the three-dimensional deformity of structural scoliosis. While his procedures appeared to be symmetrical in the coronal plane the production of a symmetrical lordosis by other workers (Wittebol 1956; Gottlieb et al. 1957; Lawton et al. 1983) failed to produce a scoliosis despite the animals being followed throughout growth. This is because rabbits have perfectly symmetrical spines and there is no inherent instability in either coronal or transverse planes to impart directional buckling to the lordosis, unlike "normal" growing children, in whom mild coronal plane curvatures can be perceived in as many as 20% (Dickson 1983). Only when a lateral "releasing" procedure was added (Lawton et al. 1983, 1985) was the lordosis

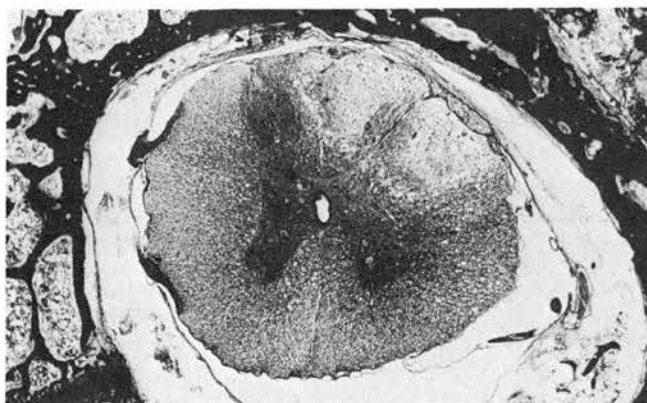


Fig. 15

Histological examination of the spinal cord from an animal with a rapidly progressive scoliosis. Transverse section through the apex of the scoliosis. (Luxol fast blue with haematoxylin and eosin, $\times 25$.)

rendered unstable and all components of the three-dimensional deformity produced with growth. Somerville (1952) therefore could have rendered his lordoses asymmetrical in the three animals that subsequently went on to develop the deformity of structural scoliosis.

The 13 animals in Group A who went on to develop a slowly progressive lordoscoliosis concave to the side of suture without additional lateral soft-tissue damage at the apex demonstrate that the three-dimensional deformity of structural scoliosis can be produced by simple biplanar mechanical means. The absence of any form of neurological damage in these animals verifies that a paralytic type of scoliosis has not been inadvertently produced. Moreover, the eight animals in Group A who lost or failed to develop a lordosis did not produce a progressive structural scoliosis and this demonstrates clearly that this model of sublaminae wiring is not in itself responsible for the deformity. The Group A animals developing the slowly progressive deformity showed no evidence of growth retardation or cardiorespiratory failure and thus represent the animal equivalent of late-onset scoliosis, which we know clinically to be a question of deformity only (Dickson 1985).

The addition of the lateral cauterising procedure in Group B produced a very rapidly progressive and serious deformity in 12 animals, but this was not for the originally hypothesised reason. The cauterisation had produced cord damage in the dorsolateral quadrant and thus added a significant paralytic component to the instability of the lordosis. The two animals in Group B who developed the slowly progressive deformity of Group A had no evidence of cord damage and thus were examples of the biplanar mechanical model. The development of cardiorespiratory failure with growth retardation indicates the danger of developing a severe spinal deformity at a young age and these are the animal equivalent of the early-onset progressive scoliosis with its notorious morbidity and mortality.

When the lateral cauterisation only was performed (Group C) the animals which went on to develop a progressive scoliosis provide a strongly suggestive link between the work of Langenskiöld and Michelsson (1961, 1962), who did not study the spinal cord, and those who have produced a progressive structural scoliosis by way of neurological damage (Liszka 1961; MacEwen 1968; Alexander et al. 1972; Taffs et al. 1979; De Salis et al. 1980; Pincott et al. 1984). Unlike the human spinal cord, the rabbit cord received its arterial supply segmentally at each level and damage to any of these segmental feeders gives rise to a cord infarct. The close proximity of the segmental feeder to rib head, transverse process and costotransverse ligament suggests that the mechanism of production of a scoliosis by all these workers has been by the production of cord damage. By contrast, when Robin (1966) divided the costotransverse ligament in baboons, which have the same sort of artery of Adamkiewicz cord supply as humans, no scoliosis was

produced. As Somerville (1952) did not look at the status of the cord, it could be that his laminar cautery either produced direct thermal damage or segmental arterial ablation, and thus the scoliosis produced may have been of the neuromuscular variety.

These studies have therefore demonstrated that a deformity very similar to late-onset idiopathic scoliosis can be produced by a simple biplanar mechanical method and that the addition of contralateral neurological damage renders the deformity produced more progressive. There is much of clinical relevance to this observation. Late-onset idiopathic scoliosis is not associated with any primary neuromuscular problem (Saartok et al. 1984; Zetterburg et al. 1984), although impairment of spinal balance mechanisms favours progression (Sahlstrand and Petruson 1979).

Thus it is progression potential rather than prevalence rate which is affected by a lessened ability to resist the rotational buckling of a fundamental lordosis. The situation is even more obvious in the infantile idiopathic progressive deformity, which is characterised by low birth weight, hypotonic, floppy babies with low neurological development scores (Mehta 1984). Moreover, Lloyd-Roberts et al. (1978) produced evidence of neurological impairment when he studied the spine of an infant with malignant progressive idiopathic scoliosis. The vast majority of infantile curves resolve spontaneously but the evidence suggests that it is impaired neurological balance mechanisms that favour malignant progression.

These studies therefore not only provide models of late-onset and early-onset structural scoliosis for further investigation, particularly of the pathogenesis of these deformities and their cardiopulmonary consequences, but also rationalise much previous confusing work on experimental scoliosis with important clinical implications.

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REFERENCES

- Adams W. *Lectures on the pathology and treatment of lateral and other forms of curvature of the spine*. London: J Churchill & Sons, 1865.
- Alexander MA, Bunch WH, Ebbesson SOE. Can experimental dorsal rhizotomy produce scoliosis? *J Bone Joint Surg [Am]* 1972;54-A:1509-13.
- Arkin AM, Simon N. Radiation scoliosis: an experimental study. *J Bone Joint Surg [Am]* 1950;32-A:396-401.
- Bisgard JD. Experimental thoracogenic scoliosis. *J Thorac Surg* 1934-5;4:435-42.
- Bisgard JD, Musselman MM. Scoliosis: its experimental production and growth correction; growth and fusion of vertebral bodies. *Surg Gynecol Obstet* 1940;70:1029-36.

- Bobechko WP.** Spinal pacemakers in scoliosis. *J Bone Joint Surg [Br]* 1973;55-B:232-3.
- De Salis J, Beguiristain JL, Cañadell J.** The production of experimental scoliosis by selective arterial ablation. *Int Orthop* 1980;3:311-5.
- Dick H.** A new instrument for the treatment of spinal curvature. *Medical Times and Gazette* 1864;II:203-4.
- Dickson RA.** Scoliosis in the community. *Br Med J* 1983;286:615-8.
- Dickson RA.** Conservative treatment for idiopathic scoliosis. *J Bone Joint Surg [Br]* 1985;67-B:176-81.
- Dickson RA, Lawton JO, Archer IA, et al.** Combined median and coronal plane asymmetry: the essential lesion of progressive idiopathic scoliosis. *J Bone Joint Surg [Br]* 1983;65-B:368.
- Dickson RA, Lawton JO, Archer IA, Butt WP.** The pathogenesis of idiopathic scoliosis: biplanar spinal asymmetry. *J Bone Joint Surg [Br]* 1984;66-B:8-15.
- Duraiswami PK.** Experimental causation of congenital skeletal defects and its significance in orthopaedic surgery. *J Bone Joint Surg [Br]* 1952;34-B:646-98.
- Engel D.** Experiments on production of spinal deformities by radium. *AJR* 1939;42:217-34.
- Gottlieb O, Jørgensen JB, Movin R.** Experimental scolioses (advance report). *Acta Orthop Scand* 1957;26:295-6.
- Haas SL.** Experimental production of scoliosis. *J Bone Joint Surg* 1939;21:963-8.
- Ingalls TH, Curley FJ.** Principles governing the genesis of congenital malformations induced in mice by hypoxia. *New Engl J Med* 1957;257:1121-7.
- Langenskiöld A, Michelsson J-E.** Experimental progressive scoliosis in the rabbit. *J Bone Joint Surg [Br]* 1961;43-B:116-20.
- Langenskiöld A, Michelsson J-E.** The pathogenesis of experimental progressive scoliosis. *Acta Orthop Scand* 1962;Suppl 59.
- Lawton JO, Butt WP, Dickson RA.** Experimental idiopathic scoliosis. *J Bone Joint Surg [Br]* 1983;65-B:657.
- Lawton JO, Scott SAJ, Dickson RA.** Physiological treatment for idiopathic scoliosis: experimental basis. *J Bone Joint Surg [Br]* 1985;67-B:160.
- Liszka O.** Spinal cord mechanisms leading to scoliosis in animal experiments. *Acta Med Pol* 1961;2:45-63.
- Lloyd-Roberts GC, Pincott JR, McMeniman P, Bayley IJL, Kendall B.** Progression in idiopathic scoliosis: a preliminary report of a possible mechanism. *J Bone Joint Surg [Br]* 1978;60-B:451-60.
- Manning CW.** Experimental scoliosis. In: Zorab PA, ed. *Proceedings of a second symposium on scoliosis: causation*. Edinburgh etc: E & S Livingstone, 1968:11-4.
- MacEwen GD.** Experimental scoliosis. In: Zorab PA, ed. *Proceedings of a second symposium on scoliosis: causation*. Edinburgh etc: E & S Livingstone, 1968:18-20.
- Mehta MH.** Infantile idiopathic scoliosis. In: Dickson RA, Bradford DS, eds. *Orthopaedics 2. Management of spinal deformities*. London etc: Butterworths, 1984:101-20.
- Michelsson J-E.** The development of spinal deformity in experimental scoliosis. *Acta Orthop Scand* 1965;Suppl 81.
- Miles M.** Vertebral changes following experimentally produced muscle imbalance: preliminary report. *Arch Phys Med* 1947;28:284-9.
- Nachlas IW, Borden JN.** Experimental scoliosis - the role of epiphysis. *Surg Gynecol Obstet* 1950;90:672-80.
- Piggott H.** Experimentally produced scoliosis in animals. In: Zorab PA, ed. *Proceedings of a second symposium on scoliosis: causation*. Edinburgh etc: E & S Livingstone, 1968:15-7.
- Pincott JR, Davies JS, Taffs LF.** Scoliosis caused by section of dorsal spinal nerve roots. *J Bone Joint Surg [Br]* 1984;66-B:27-9.
- Ponseti IV.** Skeletal lesions produced by aminonitriles. *Clin Orthop* 1957;9:131-44.
- Roaf R.** The basic anatomy of scoliosis. *J Bone Joint Surg [Br]* 1966;48-B:786-92.
- Robin GC.** Experimental paralytic scoliosis. *Israel J Med Sci* 1966;2:208-11.
- Saartok T, Dahlberg E, Bylund P, Eriksson E, Gustafsson J.** Steroid hormone receptors, protein and DNA in erector spinae muscle from scoliotic patients. *Clin Orthop* 1984;183:197-207.
- Sahlstrand T, Petruson B.** A study of labyrinthine function in patients with adolescent idiopathic scoliosis. I. An electro-nystagmographic study. *Acta Orthop Scand* 1979;50:759-69.
- Schwartzmann JR, Miles M.** Experimental production of scoliosis in rats and mice. *J Bone Joint Surg* 1945;27:59-69.
- Somerville EW.** Rotational lordosis: the development of the single curve. *J Bone Joint Surg [Br]* 1952;34-B:421-7.
- Taffs LF, Magrath DI, Lytton NA.** Monkey scoliosis. *Lancet* 1979;i:1078-9.
- Whittle MW, Evans M.** Instrument for measuring the Cobb angle in scoliosis. *Lancet* 1979;i:414.
- Wittebol P.** Idiopathic scoliosis (experimental investigation). *Arch Chir Neerl* 1956;8:269-79.
- Yamamoto H.** Experimental scoliosis in rachitic bipedal rats. *Toku-shima J Exp Med* 1966;13:1-34.
- Zetterburg C, Björk R, Örtengren R, Andersson GBJ.** Electromyography of the paravertebral muscles in idiopathic scoliosis: measurements of amplitude and spectral changes under load. *Acta Orthop Scand* 1984;55:304-9.

SPONDYLOLYSIS IN THE UPPER LUMBAR SPINE

A STUDY OF 32 PATIENTS

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Reports of spondylolysis in vertebrae other than those of the lower lumbar spine are rare. We report 32 patients with upper lumbar spondylolysis who have been studied clinically, radiologically and scintigraphically. Twenty patients had bilateral lesions, and seven of those with unilateral lesions had structural changes or anomalies in the opposite posterior arch. Positive scans were found to be associated with a short clinical history, and indicated stress-related lesions.

Our findings suggest that mechanical factors may play a role in the aetiology of spondylolysis in the upper lumbar spine similar to that which they play in the lower lumbar spine, and that local structural anomalies may contribute to abnormal loading of these vertebrae.

Spondylolysis is a common finding in the lower lumbar spine and is said to occur in from 2% to 10% of active young individuals (Roche and Rowe 1951; Raynal, Collard and Elbanna 1977). The role of mechanical stress has been emphasised in recent years as the major aetiological factor in its development in this region of the vertebral column (Wiltse, Widell and Jackson 1975; Farfan, Osteria and Lamy 1976). The vulnerability of the neural arches of the fourth and fifth lumbar vertebrae to fatigue failure after cyclic loading has also been demonstrated in the laboratory (Cyron and Hutton 1978; Patwardhan et al. 1985).

Spondylolysis in the upper lumbar spine, however, is much rarer, and has been the subject of mainly sporadic case reports (Knight, Burleson and Higginbotham 1977; Jackson, Kirwan and Sullivan 1978; Ravichandran 1981; Alcalay, Dekel and Oliver 1984). In that area mechanical stress may not provide a satisfactory explanation for the occurrence of spondylolysis, and genetic factors may be more significant (Ohta 1967).

We present a description of 32 patients with spondylolysis in the upper lumbar spine. From a study of

these patients it would appear that local structural abnormalities may account for the excessive loading of these vertebrae, so that mechanical factors may be as important in the upper as in the lower lumbar spine.

MATERIALS AND METHODS

A retrospective review was undertaken of 32 patients with spondylolysis of the first, second and third lumbar vertebrae, seen at Hadassah Hospital in Jerusalem over a period of four years. Their clinical histories were compared with their radiographic findings and with the scintigraphic data where this was available. There were 27 men and five women, with an age range from 18 to 74 years (average 27 years).

In all, there were 52 spondylolytic defects: seven lytic lesions at L1 in four patients, 13 at L2 in eight patients, and 32 at L3 in 20 patients. Twenty patients had bilateral spondylolytic defects: three at L1, five at L2 and twelve at L3. Another seven patients with unilateral spondylolysis had structural changes or anomalies in the opposite neural arch.

Eleven of the 32 patients in the series also had lytic lesions at multiple levels in the lower lumbar spine. There were a variety of additional radiographic findings at the lumbosacral junction, including seven cases of facet hypoplasia, six of spina bifida, and three of hemisacralisation. There were four patients with structural scoliosis and one with postural thoracic kyphosis.

The patients could be divided into three groups according to clinical history. In the first group were four asymptomatic patients where the pars defect was an incidental finding. In the second were 13 individuals who had suffered an acute traumatic incident, or a period of repeated but intense minor trauma (this was typical of

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CONSERVATIVE TREATMENT FOR IDIOPATHIC SCOLIOSIS.

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Why this complex three-dimensional deformity of lordo-scoliosis cannot be satisfactorily treated by any conservative method was explained using biomechanical and biological principles.

blood flow through ischaemic legs by capillary vasodilatation, thereby postponing amputation and promoting the healing of skin ulcers, and to block autonomic pain transmission. The exact action of phenol on the sympathetic chain is unknown, since histological appearances are unchanged after its use (Kester and Leveson 1981).

The accidental injection of phenol into the subarachnoid space has been reported by Smith, Davidson and Ruckley (1978) who were using a blind technique; radiological control, with an image intensifier to position the needle accurately, reduces the hazard. An intravascular injection causes only transient tinnitus and flushing (Reid, Watt and Gray 1970). The postoperative side-effects of phenol also are limited to a burning numbness in the groin for a few weeks in about 15% of cases and, occasionally, some postural hypotension.

Lower limb ischaemia affects large numbers of elderly and weak people. In 1974 Harris *et al.* reported that less than half of a series of elderly atherosclerotic patients who had amputations survived for more than six months and that the average time they spent in hospital recovering from operation was nearly two months. In the past, lumbar sympathectomy seemed to offer an attractive alternative to amputation, permanently increasing blood flow through the foot by a factor of two or three (Gillespie 1960). It was widely assumed that this increased blood flow was beneficial, but research, largely done in North America (Richards 1970; Abramson 1978; Boulton, Scarpello and Ward 1982; Lindenauer and Cronenwett 1982; Walker 1982) has demonstrated that sympathectomy does not alter blood flow in the nutritive capillaries and that the increase in total flow is due to a fall in peripheral resistance from the dilatation of arteriovenous anastomoses. These anastomoses are solely under the control of the autonomic nervous system, whereas the capillaries of muscle and skin have a high myogenic tone and respond to local levels of oxygen and nutrients.

It is ironic that the validity of lumbar sympathectomy should be questioned just when the availability of the image intensifier has made the technique of chemical lumbar sympathectomy available to the average registrar in the average hospital (Klopfer 1983). Happily, in one application sympathectomy remains unchallenged, that is in the relief of ischaemic rest pain. Of 189 men treated by Reid, Watt and Gray (1970) for ischaemic rest pain associated with trophic changes, cyanosis, oedema or ulceration, 76.7% were relieved of pain for a mean of

4.25 years and 59% had pain relief from the time of injection until they died. These figures accord well with the success rates for surgical lumbar sympathectomy in the United States and Britain in the 1960s (Richards 1970).

Moore and Walton (1981) found no difference in effectiveness between operative and chemical sympathectomy and considered that, despite the gradual fall of skin temperature and blood flow from the peaks of immediate postoperative values, the most important criterion of success was freedom from rest pain. This pain, which is constant in severe leg ischaemia, must be clearly distinguished from intermittent claudication. Phenol "neurolysis" produces no objective benefit in intermittent claudication (Fyfe and Quin 1975) and there is no sound scientific basis for its use in this condition (Lindenauer and Cronenwett 1982).

Phenol lumbar sympathectomy is a safe, effective, economical and widely available treatment for a painful condition which affects thousands of otherwise untreatable elderly people.

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REFERENCES

- Abramson DI. *Circulatory diseases of the limbs: a primer*. New York, London: Grune & Stratton, 1978.
- Boulton AJM, Scarpello JHB, Ward JD. Venous oxygenation in the diabetic neuropathic foot. Evidence of arteriovenous shunting? *Diabetologia* 1982;22:6-8.
- Fyfe T, Quin RO. Phenol sympathectomy in the treatment of intermittent claudication: a controlled trial. *Br J Surg* 1975;62: 68-71.
- Gillespie JA. Late effects of lumbar sympathectomy on blood-flow in the foot in obliterative vascular disease. *Lancet* 1960;i:891-4.
- Harris PL, Read F, Eardley A, Charlesworth D, Wakefield J, Sellwood RA. The fate of the elderly amputees. *Br J Surg* 1974;61:665-8.
- Kester RC, Leveson SH. *A practice of vascular surgery*. London: Pitman, 1981.
- Klopfer GT. Neurolytic lumbar sympathetic blockade: a modified technique. *Anaesth Intensive Care* 1983;11:43-6.
- Lindenauer SM, Cronenwett JL. What is the place of lumbar sympathectomy? *Br J Surg* 1982;69:S32-3.
- Moore PJ, Walton MR. Chemical and operative lumbar sympathectomy. *Br J Clin Pract* 1981;35(6):233-4.
- Reid W, Watt JK, Gray TG. Phenol injection of the sympathetic chain. *Br J Surg* 1970;57:45-50.
- Richards RL. *Peripheral arterial disease: a physician's approach*. Edinburgh: E & S Livingstone, 1970.
- Smith RC, Davidson NMCD, Ruckley CV. Hazard of chemical sympathectomy. *Br Med J* 1978;i:552-3.
- Walker PM. Editorial. Is there still a place for lumbar sympathectomy. *Can Med Assoc J* 1982;127(5):353-4.

CONSERVATIVE TREATMENT FOR IDIOPATHIC SCOLIOSIS

R. A. DICKSON

Because idiopathic scoliosis commences and may progress during the period of spinal growth, it has been subdivided according to when it begins (James 1954); thus infantile, juvenile and adolescent types are recognised (Goldstein and Waugh 1973). While a one-year-old baby with a 60° idiopathic thoracic curve unquestionably has infantile scoliosis, classification becomes progressively more difficult the older the child; and although a 12-year-old girl with a 90° curve is an adolescent, she does not have adolescent-onset idiopathic scoliosis, as the deformity certainly started many years earlier. Furthermore, there is no clear evidence that juvenile-onset idiopathic scoliosis exists. Of James' 134 patients with thoracic scoliosis, only 16 were tentatively classified as of juvenile onset and he did not think these worth separating from the infantile group (James 1954). Such cases may well be a hangover from infancy (Mehta 1977). There is much merit therefore in considering only two categories, early-onset and late-onset (Ponseti and Friedman 1950; Figs 1 to 4). The prevalence rate, natural history and the consequences of untreated scoliosis, as well as the strategy for treatment and its efficacy, differ very considerably between early and late-onset types. Treatment for the more common and more benign late-onset case is more "standard" and will therefore be discussed first.

LATE-ONSET IDIOPATHIC SCOLIOSIS

The need for treatment. The strategy for treating idiopathic scoliosis depends principally upon the size of the deformity and its potential for progression. If the deformity is acceptable at presentation, then preservation of acceptability is the aim; this is the place of conservative management. If the deformity is unacceptable, then the objective must be to make it acceptable and keep it so; this is the aim of surgical management. In order to decide if idiopathic scoliosis needs treatment at all, the consequences of leaving it untreated must be known. It is, of course, known that scoliosis can cause significant disability, with economic implications (Dahlberg and

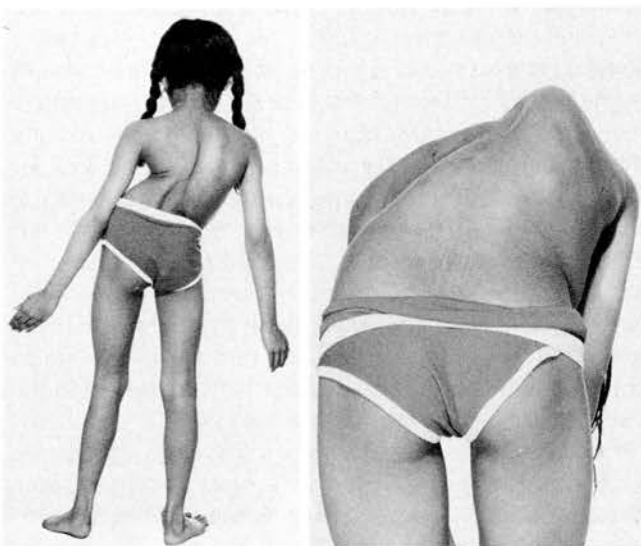


Fig. 1

Fig. 2

Early-onset idiopathic scoliosis—a very serious condition producing cardiopulmonary compromise and horrendous deformity. Figure 1—Erect. Figure 2—Close-up in the forward-bending position.

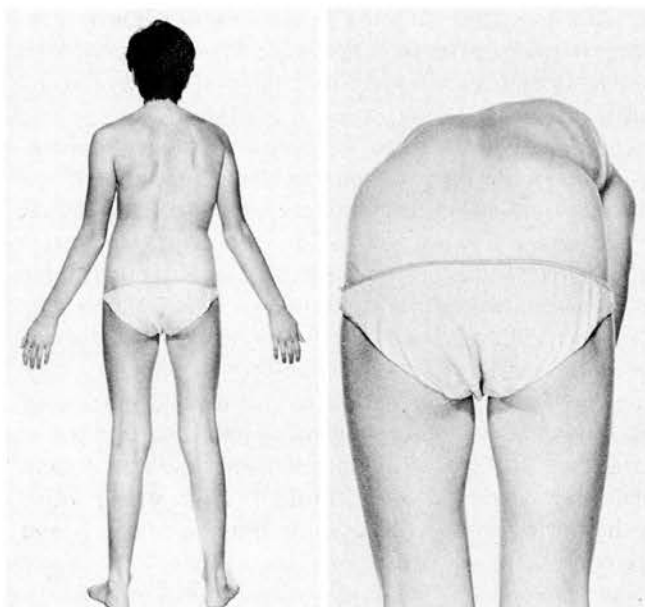


Fig. 3

Fig. 4

Late-onset idiopathic scoliosis—a problem of deformity only. Figure 3—Erect. Figure 4—Close-up in the forward-bending position.

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Nachemson 1977), and that the cardiopulmonary complications can be a source of morbidity and mortality (Nachemson 1968; Nilsonne and Lundgren 1968; Collis and Ponseti 1969). But this applies only to early-onset cases; and then only to severe ones, for example, a thoracic curve of over 60° by the age of seven or eight years, when the pulmonary parenchyma is developing (Davies and Reid 1971).

In contradistinction there are no obvious organic consequences of late-onset idiopathic scoliosis, even if the deformity exceeds 100° (Kostuik, Israel and Hall 1973; Ponder *et al.* 1975; Dickson and Leatherman 1976). Late-onset idiopathic scoliosis is a problem of deformity only. The 52 late-onset idiopathic cases in Nachemson's original study of 130 patients fared no differently from their straight-backed counterparts as regards organic health (Nachemson 1968). With regard to the deformity, it is of course true that the bigger the deformity the greater the likelihood of social and psychological implications (Nilsonne and Lundgren 1968; Bengtsson *et al.* 1974). The patient's opinion on this subject clearly matters more than that of the surgeon.

Natural history. As there has never been a controlled trial of conservative treatment its efficacy can only be determined by evaluating it against the little we know of the natural history of the late-onset curve. There are two quite different sources of information. Early studies of selected groups of children who presented to scoliosis clinics suggested a considerable progression potential if the onset was under 10 years of age or before the menarche (Risser and Ferguson 1936; Ponseti and Friedman 1950; James 1954; Heine and Reher 1975). More recent data come from those school screening programmes which have included a longitudinal survey (Brooks *et al.* 1975; Rogala, Drummond and Gurr 1978; Dickson *et al.* 1980; Dickson 1983). When children with non-structural curves are excluded, only 10% show evidence of progression, whereas twice as many improve and more than two-thirds remain static. The greatest progression potential is associated with the young girl who has a right thoracic curve, but she represents less than one in a thousand of those screened. The difference between the data from these two sources, although difficult to interpret (Leaver, Alvik and Warren 1982), suggests a change to a more benign natural history, and this is supported by the observation that where a lot of screening has been performed the need for both conservative and operative management has been much less (Lonstein *et al.* 1982). Conservative treatment should then be set against this background. Three conservative methods need to be discussed: bracing, casting and electrospinal stimulation.

Brace treatment. While various contraptions for the scoliotic spine have been used since the time of Hippocrates, real enthusiasm for conservative treatment started with the Milwaukee brace (Blount and Schmidt

1957; Blount 1958). Newer technology refers to this brace as a CTLSO (cervical-thoracic-lumbar-sacral orthosis) (Nash 1980). Although the brace was not primarily intended for the conservative management of idiopathic scoliosis, it was soon used for that purpose (Blount 1972; Moe 1973). Early mechanical studies suggested that the brace might function by exerting distraction between the head and the pelvis (Schultz and Galante 1969; Galante *et al.* 1970) and this mode of action was corroborated by the harmful effects on dentition thereby produced (Alexander 1966). A change to the throat mould type of brace led to a great reduction in the distraction force with no obvious dental problems (Northway, Alexander and Riolo 1974); and a change in biomechanical approach led to three-point fixation with particular emphasis placed on the localiser pad (Andriacchi *et al.* 1976).

Without a clear understanding of the three-dimensional nature of the deformity it would be tempting to think that the brace might work in the manner described, but this is not so. The primary deformity of idiopathic scoliosis is a lordosis at the curve apex (Adams 1882; Somerville 1952; Roaf 1966; Dickson *et al.* 1983, 1984) and it is rotation of this lordosis to the side which produces the secondary scoliotic deformity. An ideal of conservative treatment would then be to recreate the normal spinal shape in the sagittal plane; this, however, would imply flexion, which enhances rotation and produces an increase in the secondary scoliotic deformity. In contradistinction, the opposite deformity, the kyphosis of Scheuermann's disease, is ideally suited to conservative management, because the deformity is rotationally stable and braces which cause spinal extension produce a true physiological correction of the deformity (Bradford *et al.* 1974). The brace is capable, however, of producing a small temporary corrective effect in idiopathic scoliosis. Blount stressed the need for obliteration of the lumbar lordosis in the brace (Blount and Moe 1973) and this produces thoracic extension above. There is now more room for the thoracic lordosis to be accommodated with a derotation effect, but at the possible expense of increasing the primary lordosis (Winter, Lovell and Moe 1975; Figs 5 and 6). The other important effect of the brace is to splint the spine to the pelvis in the erect position which thus prevents the harmful effect of flexion.

With this mode of action it is not surprising to find that the optimal result of brace wearing is when the curve measures exactly the same at the end of treatment as it did at the beginning (Keiser and Shufflebarger 1976; Edmondson and Morris 1977; Mellencamp, Blount and Anderson 1977; Tolo and Gillespie 1978; Blount 1981). These studies also suggest that the more the curve has progressed beyond 30° before the commencement of treatment, the less satisfactorily can curve progression be attenuated, as gravity and the rigidity of the secondary deformities more successfully defeat the intentions of



Fig. 5



Fig. 6

The effect of obliteration of the lumbar lordosis. Figure 5—A thoracic deformity associated with a lumbar lordosis viewed from above. Figure 6—The same deformity viewed from above with the lumbar lordosis obliterated. Marked derotation has occurred.

treatment. The best results of brace treatment are therefore achieved with smaller curves; their natural history, however, demonstrates that very few would have progressed if left untreated.

The brace is supposed to be worn for 23 hours out of 24, although it would appear to be unnecessary at night when repeated cycles of spinal flexion are not usually performed. Although exercises have no corrective effect on idiopathic curves (Stone *et al.* 1979), an exercise programme is prescribed for the one hour a day spent out of the brace lest the spine become unduly stiff; this programme should not, however, include flexion exercises as these will undo what the brace has been trying to achieve for the previous 23 hours.

Set against the background of natural history there is no evidence that Milwaukee brace treatment alters the course of the scoliosis. This is a very serious matter, as countless numbers of children may have endured brace treatment for no detectable benefit. If the effect of the brace on girls with progressive thoracic curves was not diluted by the inclusion of curves at other sites with little or no progression potential, then the lumbar lordosis-obliterating and flexion-preventing effect of the brace ought to prevent progression (Dickson *et al.* 1984). Cognisant of this problem the British Orthopaedic Association and the British Scoliosis Society (1983) are right to stress the need for carefully controlled studies of idiopathic scoliosis, preferably throughout life.

It recently became apparent that low thoracic and lumbar curves did not require the full superstructure of a

Milwaukee brace (Park *et al.* 1977; Watts, Hall and Stanish 1977; Winter and Carlson 1977); this is because flexion of these low curves can be prevented even by an underarm brace, TLSO (thoracic-lumbar-sacral orthosis). But the other mode of action, obliteration of the lumbar lordosis, is more obvious and produces a bigger temporary corrective effect. There are no controlled trials with an underarm brace and the follow-up is much shorter than with a Milwaukee brace; consequently their efficacy also is questionable, though of course their lighter weight and smaller size make them more acceptable to the patient.

Another problem concerns the duration of spinal growth. The only period after the intra-uterine phase when growth velocity increases is during the adolescent growth spurt, which is maximal at about the age of 12 years in girls and 14 in boys (Scammon 1927). While idiopathic curves are particularly liable to deteriorate during this phase, general skeletal maturity is reached two years later (Tanner 1962). The conventional time when the patient is weaned from the brace has been when the iliac crest and vertebral ring apophyses fuse (Risser and Ferguson 1936; James 1954; Risser 1964). It is well known, however, that spinal growth continues for a further 10 years until the vertebral epiphyses are fused and that the vertebral apophyses have nothing whatever to do with spinal growth nor does their fusion indicate cessation of growth (Bick, Copel and Spector 1950; Bick and Copel 1951; Inkster 1951; Calvo 1957; Tupman 1962; Larsen and Nordentoft 1962; Bernick and Caillet 1982). Recent studies of idiopathic curves beyond general skeletal maturity do, in fact, demonstrate progression in the majority of cases (Hassan and Bjerkreim 1983; Weinstein and Ponseti 1983). While these studies have suggested that the effect of pregnancy on ligaments might be a responsible factor, it ought not to be forgotten that, in young women, the spine is still growing. Even if there was evidence that the brace did prevent progression, treatment would need to be continued for much longer than the patient would tolerate.

Cast management. The pioneers of the treatment of scoliosis obtained correction by using plaster casts (Risser *et al.* 1953; Risser 1955) and it was remarkable to what good use they put them (Moe and Valuska 1966). With the advent of the Milwaukee brace, enthusiasm for plaster in the conservative treatment rapidly waned in many parts of the world. French surgeons, however, did not lose their faith in plaster techniques and have developed the EDF (elongation-derotation-flexion) cast as an alternative to brace treatment (Cotrel and Morel 1964). The function of this cast is precisely that of the brace, with obliteration of the lumbar lordosis and elimination of spinal flexion as the two priorities. Each cast is worn for three or four months until its wear or the patient's growth indicates that a new one is required. The patients cannot bathe, but it is extraordinary how easy it is to change the inner vest and the underwear while the

cast is in place. Furthermore, the cast has a window on the concave side posteriorly and one on the convex side anteriorly; these facilitate derotation exercises and allow pressure pads or balloons to be inserted over the rotational prominences. Since the object of conservative treatment is to finish up with the least deformed torso, "bracers" would do well to compare their end-results with French "casters" although, as with the majority of interesting questions, the answer has never been elucidated by a controlled study. Between casts a programme of non-skeletal traction and exercises is carried out (Cotrel and D'Amore 1968). This is of no benefit, however, in terms of curve correction (Nachemson and Nordwell 1977; Dickson and Leatherman 1978). Indeed, traction of any kind provides no real correction of curves of any magnitude, only moving each curve through its natural range of flexibility (Edgar, Chapman and Glasgow 1982).

Electrospinal stimulation. Recently, attention has been directed towards obtaining temporary correction of the scoliosis by electrical stimulation of the spinal musculature on the convexity of the curve (Bobechko 1974). Like the development of orthotic and cast treatment, electrical stimulation focuses on the secondary coronal-plane deformity and only moves the spine within its natural range of elasticity. Furthermore, electrical stimulation stemmed from the belief that there was a neuromuscular basis to the deformity, a belief which is unlikely to be substantiated (Dickson *et al.* 1984).

The fact that mild coronal-plane curvatures in animals can be produced by stimulating muscles on one side (Olsen *et al.* 1975; Monticelli *et al.* 1975; Bobechko, Herbert and Friedman 1976) is not surprising. This is precisely what happens when someone with a straight spine bends to one side and then resumes the erect position. Some improvement in curve magnitude has been demonstrated during convex muscle stimulation (Bobechko, Herbert and Friedman 1979); this demonstrates the innate flexibility that the mild idiopathic curve enjoys, but there is no evidence that any real correction follows electrospinal stimulation (Axelgaard and Brown 1983). Again it is the rotationally unstable nature of the primary lordotic deformity which militates against effective conservative treatment; with the uniplanar and rotationally stable kyphotic deformity, however, electrical surface stimulation can, like the brace or cast, give rise to permanent correction (Axelgaard, Brown and Swank 1982).

EARLY-ONSET IDIOPATHIC SCOLIOSIS

Natural history. This fascinating condition, first reported from Holland (Harrenstein 1929), tends to affect children from birth to three years of age. Boys are affected more commonly than girls and thoracic curves are more frequently convex to the left (James 1951; James, Lloyd-Roberts and Pilcher 1959; Lloyd-Roberts and Pilcher 1965; Wynne-Davies 1975; Thompson and Bentley

1980). Here progression potential is particularly relevant. It was first thought that the condition could be divided into two types—progressive and resolving—depending upon the size of the rib-vertebra angle difference (RVAD; Mehta 1972). The picture is not so clear, however, and three types are now recognised—progressive, static and resolving (Mehta 1977). While an RVAD of less than 20° which then reduces in magnitude confidently diagnoses the resolving curve, an RVAD in excess of 20° or one that is increasing does not necessarily imply a progressive curve. Other factors also are important. Thoracic and thoracolumbar curves and small initial curves (Mehta 1977; Thompson and Bentley 1980) tend to resolve, while double structural curves have a definite progression potential (Ceballos *et al.* 1980). The most serious progression appears to occur in the hypotonic, low birth weight baby in whom the condition has been referred to as "malignant" idiopathic scoliosis (Mehta 1977).

A very interesting trend has emerged over the last 35 years. Early reports indicated a great preponderance of the progressive type of curve (James 1951; Scott and Morgan 1955; James *et al.* 1959), but this situation then changed dramatically and the last 20 years has seen a marked reversal of the proportions with 90% or more resolving (Lloyd-Roberts and Pilcher 1965; Mau 1968; Thompson and Bentley 1980; Ceballos *et al.* 1980). The incidence of these infantile curves also has rapidly declined and the condition is now rare; whether or not this decline is due to prone lying in the cot is unclear (McMaster 1983). These changes in the natural history of early-onset progressive idiopathic scoliosis are very welcome, as these are the curves associated with serious cardiopulmonary disease at an early age, and they also develop horrifying deformities.

Conservative treatment. When malignant progressive curves were more common, treatment presented great problems. Progression potential was far too great to be attenuated by a Milwaukee brace (James *et al.* 1959), but posterior fusion was withheld for as long as possible in order to avoid increasing the primary lordosis; meanwhile the deformity progressed inexorably in the brace. By the time posterior fusion was performed, the deformity was often too far advanced for treatment; moreover, there is no clear evidence that fusion reduced the rate of subsequent progression (Letts and Bobechko 1974; McMaster and Macnicol 1979).

Unlike late-onset deformities there is some evidence that early-onset idiopathic scoliosis can be treated conservatively. Mehta, who has contributed much to our knowledge of infantile idiopathic scoliosis, recognised early the bad prognosis associated with the hypotonic infant, and the moment she saw such a child she applied an elongation-rotation-flexion (EDF) cast (Mehta and Morel 1979). Surprisingly, the occasional case that had all the ingredients for rapid progression appeared to become static, or even to resolve, and the RVAD became

smaller or did not increase. This perhaps demonstrates the effect of obliteration of the lumbar lordosis and the prevention of flexion in these very supple spines; but the cast must also have allowed the thoracic spine to become naturally kyphotic in those that subsequently resolved.

There are, clearly, two important aspects of the conservative management of the early-onset case, namely, prevention and casting. Although debate continues as to whether the deformity is due to intra-uterine moulding (Browne 1936) or to positioning in the cot (Mau 1968), prone lying does appear to have an inhibitory effect; it must be insisted upon, particularly for the hypotonic infant (McMaster 1983). For the rare case that does develop an idiopathic curve with all the hallmarks of progression, serial EDF casts should be applied without delay (Mehta 1977).

REFERENCES

- Adams W. *Lectures on the pathology and treatment of lateral and other forms of curvature of the spine*. London: J & A Churchill & Sons, 1882.
- Alexander RG. The effects on tooth position and maxillofacial vertical growth during treatment of scoliosis with the Milwaukee brace. *Am J Orthod* 1966;**52**:161-89.
- Andriacchi TP, Schultz AB, Belytschko TB, DeWald RL. Milwaukee brace correction of idiopathic scoliosis: a biomechanical analysis and a retrospective study. *J Bone Joint Surg [Am]* 1976;**58-A**:806-15.
- Axelgaard J, Brown JC. Lateral electrical surface stimulation for the treatment of progressive idiopathic scoliosis. *Spine* 1983;**8**(3):242-60.
- Axelgaard J, Brown JC, Swank SM. Kyphosis treatment by electrical surface stimulation. *Orthop Trans* 1982;**6**:1.
- Bengtsson G, Fällström K, Jansson B, Nachemson A. A psychological and psychiatric investigation of the adjustment of female scoliosis patients. *Acta Psychiatr Scand* 1974;**50**:50-9.
- Bernick S, Cailliet R. Vertebral end-plate changes with aging of the human vertebrae. *Spine* 1982;**7**(2):97-102.
- Bick EM, Copel JW. The ring apophysis of the human vertebra. Contribution to human osteogeny. II. *J Bone Joint Surg [Am]* 1951;**33-A**:783-7.
- Bick EM, Copel JW, Spector S. Longitudinal growth of the human vertebra: a contribution to human osteogeny. *J Bone Joint Surg [Am]* 1950;**32-A**:803-14.
- Blount WP. Scoliosis and the Milwaukee brace. *Bull Hosp Joint Dis* 1958;**19**:152-65.
- Blount WP. Use of the Milwaukee brace. *Orthop Clin North Am* 1972;**3**:3-16.
- Blount WP. Editorial. The virtue of early treatment of idiopathic scoliosis. *J Bone Joint Surg [Am]* 1981;**63-A**:335-6.
- Blount WP, Moe JH. *The Milwaukee brace*. Baltimore: Williams & Wilkins, 1973.
- Blount WP, Schmidt AC. The Milwaukee brace in the treatment of scoliosis. *J Bone Joint Surg [Am]* 1957;**39-A**:693.
- Bobechko WP. Scoliosis spinal pacemakers. *J Bone Joint Surg [Am]* 1974;**56-A**:442.
- Bobechko WP, Herbert M, Friedman H. Electro-spinal instrumentation. *J Bone Joint Surg [Am]* 1976;**58-A**:156.
- Bobechko WP, Herbert M, Friedman HG. Electrospondyl instrumentation for scoliosis: current status. *Orthop Clin North Am* 1979;**10**(4):927-41.
- Bradford DS, Moe JH, Montalvo FJ, Winter RB. Scheuermann's kyphosis and roundback deformity: results of Milwaukee brace treatment. *J Bone Joint Surg [Am]* 1974;**56-A**:740-58.
- British Orthopaedic Association and the British Scoliosis Society. School screening for scoliosis. *Brit Med J* 1983;**287**:963-4.
- Brooks HL, Azen SP, Gerberg E, Brooks R, Chan L. Scoliosis: a prospective epidemiological study. *J Bone Joint Surg [Am]* 1975;**57-A**:968-72.
- Browne D. Congenital deformities of mechanical origin. *Proc R Soc Med* 1936;**29**:1409-31.
- Calvo IJ. Observations on the growth of the female adolescent spine and its relation to scoliosis. *Clin Orthop* 1957;**10**:40-7.
- Ceballos T, Ferrer-Torrelles M, Castillo F, Fernandez-Paredes E. Prognosis in infantile idiopathic scoliosis. *J Bone Joint Surg [Am]* 1980;**62-A**:863-75.
- Collis DK, Ponseti IV. Long-term follow-up of patients with idiopathic scoliosis not treated surgically. *J Bone Joint Surg [Am]* 1969;**51-A**:425-45.
- Cotrel Y, D'Amore M. Spinal traction in scoliosis. In: Zorab PA, ed. *Proceedings of a second symposium on scoliosis: causation*. Edinburgh and London: E & S Livingstone, 1968;37-43.
- Cotrel Y, Morel G. La technique de l'E.D.F dans la correction des scolioses. *Rev Chir Orthop* 1964;**50**:59-75.
- Dahlberg L, Nachemson AL. The economic aspects of scoliosis treatment. In: Zorab PA, ed. *Scoliosis: proceedings of a fifth symposium held at the Cardiothoracic Institute, Brompton Hospital, London, on 21st, 22nd September, 1976*. London: Academic Press, 1977;73-101.
- Davies G, Reid L. Effect of scoliosis on growth of alveoli and pulmonary arteries and on right ventricle. *Arch Dis Child* 1971;**46**:623-32.
- Dickson RA. Scoliosis in the community. *Br Med J* 1983;**286**:615-8.
- Dickson RA, Lawton JO, Archer IA, Butt WP. The pathogenesis of idiopathic scoliosis. Biplanar spinal asymmetry. *J Bone Joint Surg [Br]* 1984;**66-B**:8-15.
- Dickson RA, Lawton JO, Archer IA, et al. Combined median and coronal plane asymmetry: the essential lesion of idiopathic scoliosis. *J Bone Joint Surg [Br]* 1983;**65-B**:368.
- Dickson RA, Leatherman KD. Spinal deformity in adults: changing concepts. *J Bone Joint Surg [Am]* 1976;**58-A**:729.
- Dickson RA, Leatherman KD. Cotrel traction, exercises, casting in the treatment of idiopathic scoliosis: a pilot study and prospective randomized controlled clinical trial. *Acta Orthop Scand* 1978;**49**:46-8.
- Dickson RA, Stamper P, Sharp A-M, Harker P. School screening for scoliosis: cohort study of clinical course. *Br Med J* 1980;**281**:265-7.
- Edgar MA, Chapman RH, Glasgow MMS. Pre-operative correction in adolescent idiopathic scoliosis. *J Bone Joint Surg [Br]* 1982;**64-B**:530-5.
- Edmondson AS, Morris JT. Follow-up study of Milwaukee brace treatment in patients with idiopathic scoliosis. *Clin Orthop* 1977;**126**:58-61.
- Galante J, Schultz A, DeWald RL, Ray RD. Forces acting in the Milwaukee brace on patients undergoing treatment for idiopathic scoliosis. *J Bone Joint Surg [Am]* 1970;**52-A**:498-506.
- Goldstein LA, Waugh TR. Classification and terminology of scoliosis. *Clin Orthop* 1973;**93**:10-22.
- Harrenstein RJ. Die Skoliose bei Säuglingen und ihre Behandlung. *Z Orthop Chir* 1929;**52**:1-40.
- Hassan I, Bjerkreim I. Progression in idiopathic scoliosis after conservative treatment. *Acta Orthop Scand* 1983;**54**:88-90.
- Heine J, Reher H. Die Progredienz der unbehandelten idiopathischen Skoliose bis Wachstumsabschluss. *Z Orthop* 1975;**113**:87-96.
- Inkster RG. Osteology. In: Brash JC, ed. *Cunningham's text-book of anatomy*. 9th edn. London: Oxford University Press, 1951;105-331.
- James JIP. Two curve patterns in idiopathic structural scoliosis. *J Bone Joint Surg [Br]* 1951;**33-B**:399-406.
- James JIP. Idiopathic scoliosis: the prognosis, diagnosis and operative indications related to curve patterns and the age at onset. *J Bone Joint Surg [Br]* 1954;**36-B**:36-49.
- James JIP, Lloyd-Roberts GC, Pilcher MF. Infantile structural scoliosis. *J Bone Joint Surg [Br]* 1959;**41-B**:719-35.
- Keiser RP, Shufflebarger HL. The Milwaukee brace in idiopathic scoliosis: evaluation of 123 completed cases. *Clin Orthop* 1976;**118**:19-24.
- Kostuik JP, Israel J, Hall JE. Scoliosis surgery in adults. *Clin Orthop* 1973;**93**:225-34.
- Larsen EH, Nordentoft EL. Growth of the epiphyses and vertebra. *Acta Orthop Scand* 1962;**32**:210-7.
- Leaver JM, Alvik A, Warren MD. Prescriptive screening for adolescent idiopathic scoliosis: a review of the evidence. *Int J Epidemiol* 1982;**11**(2):101-11.

- Letts RM, Bobechko WP. Fusion of the scoliotic spine in young children: effects on prognosis and growth. *Clin Orthop* 1974; **101**:136-45.
- Lloyd-Roberts GC, Pilcher MF. Structural idiopathic scoliosis in infancy: a study of the natural history of 100 patients. *J Bone Joint Surg [Br]* 1965; **47-B**:520-3.
- Lonstein JE, Bjorklund S, Wanning MH, Nelson RP. Voluntary school screening for scoliosis in Minnesota. *J Bone Joint Surg [Am]* 1982; **64-A**:481-8.
- McMaster MJ. Infantile idiopathic scoliosis: can it be prevented? *J Bone Joint Surg [Br]* 1983; **65-B**:612-7.
- McMaster MJ, Macnicol MF. The management of progressive infantile idiopathic scoliosis. *J Bone Joint Surg [Br]* 1979; **61-B**:36-42.
- Mau H. Does infantile scoliosis require treatment? *J Bone Joint Surg [Br]* 1968; **50-B**:881.
- Mehta MH. The rib-vertebra angle in the early diagnosis between resolving and progressive infantile scoliosis. *J Bone Joint Surg [Br]* 1972; **54-B**:230-43.
- Mehta MH. The natural history of infantile idiopathic scoliosis. In: Zorab PA, ed. *Scoliosis: proceedings of a fifth symposium held at the Cardiothoracic Institute, Brompton Hospital, London, on 21st, 22nd September, 1976*. London: Academic Press, 1977:103-22.
- Mehta MH, Morel G. The non-operative treatment of infantile idiopathic scoliosis. In: Zorab PA, Siegler D, eds. *Scoliosis 1979*. Based on the proceedings of the sixth symposium on scoliosis held at the Cardiothoracic Institute, Brompton Hospital, London, on September 17 and 18, 1979. London: Academic Press, 1980:71-84.
- Mellencamp DD, Blount WP, Anderson AJ. Milwaukee brace treatment of idiopathic scoliosis: late results. *Clin Orthop* 1977; **126**:47-57.
- Moe JH. Indications for Milwaukee brace non-operative treatment in idiopathic scoliosis. *Clin Orthop* 1973; **93**:38-43.
- Moe JH, Valuska J. Evaluation of treatment of scoliosis by Harrington instrumentation. *J Bone Joint Surg [Am]* 1966; **48-A**:1656-7.
- Monticelli G, Ascani E, Salsano V, Salsano A. Experimental scoliosis induced by prolonged minimal electrical stimulation in the paravertebral muscles. *Ital J Orthop Traumatol* 1975; **1**:39-54.
- Nachemson A. A long term follow-up study of non-treated scoliosis. *Acta Orthop Scand* 1968; **39**:466-76.
- Nachemson A, Nordwell A. Effectiveness of preoperative Cotrel traction for correction of idiopathic scoliosis. *J Bone Joint Surg [Am]* 1977; **59-A**:504-8.
- Nash CL Jr. Current concepts review: scoliosis bracing. *J Bone Joint Surg [Am]* 1980; **62-A**:848-52.
- Nilsson U, Lundgren K-D. Long-term prognosis in idiopathic scoliosis. *Acta Orthop Scand* 1968; **39**:456-65.
- Northway RO Jr, Alexander RG, Riolo ML. A cephalometric evaluation of the old Milwaukee brace and the modified Milwaukee brace in relation to the normal growing child. *Am J Orthod* 1974; **65**:341-63.
- Olsen GA, Rosen H, Stoll S, Brown G. The use of muscle stimulation for inducing scoliotic curves: a preliminary report. *Clin Orthop* 1975; **113**:198-211.
- Park J, Houtkin S, Grossman J, Levine DB. A modified brace (Pichyl) for scoliosis. *Clin Orthop* 1977; **126**:67-73.
- Ponder CR, Dickson JH, Harrington PR, Erwin WD. Results of Harrington instrumentation and fusion in the adult idiopathic scoliosis patient. *J Bone Joint Surg [Am]* 1975; **57-A**:797-801.
- Ponseti IV, Friedman B. Prognosis in idiopathic scoliosis. *J Bone Joint Surg [Am]* 1950; **32-A**:381-95.
- Risser JC. Scoliosis: the application of body casts for the correction of scoliosis. *Am Acad Orthop Surg Instruct Course Lect* 1955; **12**:255-9.
- Risser JC. Scoliosis: past and present. *J Bone Joint Surg [Am]* 1964; **46-A**:167-99.
- Risser JC, Ferguson AB. Scoliosis: its prognosis. *J Bone Joint Surg* 1936; **18**:667-70.
- Risser JC, Lauder CH, Norquist DM, Craig WA. Three types of body casts. *Am Acad Orthop Surg Instruct Course Lect* 1953; **10**:131-42.
- Roaf R. The basic anatomy of scoliosis. *J Bone Joint Surg [Br]* 1966; **48-B**:786-92.
- Rogala EG, Drummond DS, Gurr J. Scoliosis: incidence and natural history: a prospective epidemiological study. *J Bone Joint Surg [Am]* 1978; **60-A**:173-6.
- Scammon RE. The first serial study of human growth. *Am J Phys Anthropol* 1927; **10**:329-36.
- Schultz AB, Galante JO. Measurement of forces exerted in the correction of idiopathic scoliosis using three-component dynamometers. *Exp Mech* 1969; **9**:419-24.
- Scott JC, Morgan TH. The natural history and prognosis of infantile idiopathic scoliosis. *J Bone Joint Surg [Br]* 1955; **37-B**:400-13.
- Somerville EW. Rotational lordosis: the development of the single curve. *J Bone Joint Surg [Br]* 1952; **34-B**:421-7.
- Stone B, Beekman C, Hall V, Guess V, Brooks HL. The effect of an exercise program on change in curve in adolescents with minimal idiopathic scoliosis: a preliminary study. *Phys Ther* 1979; **59**:759-63.
- Tanner JM. *Growth at adolescence with a general consideration of the effects of hereditary and environmental factors upon growth and maturation from birth to maturity*. 2nd edn. Oxford: Blackwell Scientific Publications, 1962.
- Thompson SK, Bentley G. Prognosis in infantile idiopathic scoliosis. *J Bone Joint Surg [Br]* 1980; **62-B**:151-4.
- Tolo VT, Gillespie R. The characteristics of juvenile idiopathic scoliosis and results of its treatment. *J Bone Joint Surg [Br]* 1978; **60-B**:181-8.
- Tupman GS. A study of bone growth in normal children and its relationship to skeletal maturation. *J Bone Joint Surg [Br]* 1962; **44-B**:42-67.
- Watts HG, Hall JE, Stanish W. The Boston brace system for the treatment of low thoracic and lumbar scoliosis by the use of a girdle without superstructure. *Clin Orthop* 1977; **126**:87-92.
- Weinstein SL, Ponseti IV. Curve progression in idiopathic scoliosis. *J Bone Joint Surg [Am]* 1983; **65-A**:447-55.
- Winter RB, Lovell WW, Moe JH. Excessive thoracic lordosis and loss of pulmonary function in patients with idiopathic scoliosis. *J Bone Joint Surg [Am]* 1975; **57-A**:972-7.
- Winter RB, Carlson JM. Modern orthotics for spinal deformities. *Clin Orthop* 1977; **126**:74-86.
- Wynne-Davies R. Infantile idiopathic scoliosis: causative factors, particularly in the first six months of life. *J Bone Joint Surg [Br]* 1975; **57-B**:138-41.

IDIOPATHIC SCOLIOSIS

PREVALENCE AND ETHNIC DISTRIBUTION IN SINGAPORE SCHOOLCHILDREN

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Routine examination for spinal deformity as part of a school health screening programme was introduced in Singapore in 1981. The three different ethnic groups included in the study provided figures for the prevalence of idiopathic scoliosis in an Asian population.

A three-tier system of examination was used and a total of 110 744 children in three age groups were studied. In those aged 6 to 7 years the prevalence was 0.12%. The prevalence in those aged 11 to 12 years was 1.7% for girls and 0.4% for boys, a ratio of 3.2 to 1. In girls aged 16 to 17 years the prevalence was 3.1%. In the latter two age groups there was a significantly higher prevalence in Chinese girls as compared with Malay and Indian girls. The optimal age for school screening seemed to be 11 to 12 years, but repeated examinations may be worthwhile.

In the last two decades the conservative and operative management of progressive structural scoliosis has greatly improved. If conservative treatment is started early the number of operations can be reduced (Lonstein *et al.* 1976; Torell, Nordwall and Nachemson 1981). With this in view, screening at school for the early detection of scoliosis has been introduced in many countries (Segil 1974; Brooks *et al.* 1975; Span, Robin and Makin 1976; Ascani, Salsano and Giglio 1977; Rogala, Drummond and Gurr 1978; Drummond, Rogala and Gurr 1979; Takemitsu *et al.* 1980; Dickson *et al.* 1980; Torell *et al.* 1981). In the USA, such screening is mandatory in nine states (Lonstein *et al.* 1982) and is done to some extent in others.

Shands and Eisberg (1955) studied 50 000 minifilms of the chest in the State of Delaware and detected scoliosis of at least 10° in 1.9% of the population over 14 years of age. Except for the high rates reported by Grant *et al.* in 1973 (13.4%) and Brooks *et al.* in 1975 (13.6%), the reported prevalence of scoliosis varies from 1.6% to 4.6% because of the use of different criteria or of variation in sampling (Segil 1974; Brooks *et al.* 1975; Golomb and Taylor 1975; Span *et al.* 1976; Lonstein 1977; Rogala *et al.* 1978; Drummond *et al.* 1979; Smyrnis *et al.* 1979, 1980).

The effective School Health Service in Singapore

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and the presence of three different ethnic groups (Chinese, Indian and Malay) have provided an opportunity to study the prevalence of idiopathic scoliosis in an Asian population.

MATERIAL AND METHODS

The forward-bending test for spinal deformity was introduced as part of a school health screening programme in 1981 by the University Department of Orthopaedic Surgery, Singapore General Hospital, in collaboration with the School Health Services. Moiré topography, or contourgraphy, was added to this programme in 1982.

A three-tier system similar to those described by Hensinger *et al.* (1975) and Owen *et al.* (1980) was adopted. Initial screening is done in schools by nurses who have been taught the forward-bending test. Children with a positive test are referred to the second tier, and are examined by School Health Medical Officers. A moiré photograph is taken. Some children may be discharged from review at this level, while others are referred to the third tier, a specialist clinic at the Singapore General Hospital. In this clinic a comprehensive examination is done and a standing radiograph of the spine is taken.

In 1982 three age groups of children were chosen for a study to discover the optimal age for screening. These groups were: 6 to 7 years (primary one), 11 to 12 years (primary six), and 16 to 17 years (secondary four). The last group contained only girls, since the boys had left for National Service. Altogether 110 744 children were examined, 60 167 girls and 50 577 boys.

The criteria for the diagnosis of scoliosis were a rotational prominence detected on forward bending, and a curve showing a Cobb angle of 5° or more measured on a standing radiograph.

THE SURGICAL MANAGEMENT OF IDIOPATHIC THORACIC SCOLIOSIS.

Dickson RA.

Journal of Orthopaedic Surgical Techniques; 1: 23-28, 1985.

Hitherto the standard method of dealing with an idiopathic thoracic deformity surgically was by posterior fusion using instrumentation which attempted to stretch the deformity. This was performed through a posterior spinal approach and, because the essential lesion of idiopathic thoracic scoliosis is a lordosis, the back of the spine can be tethered further by the bony fusion allowing progressive buckling of the anterior vertebral bodies. The unpredictable nature of standard treatment was therefore explained and the concept of segmental derotation of the spine while at the same time recreating the thoracic kyphosis demonstrated. The bigger the curve, the bigger the underlying degree of lordosis and the more the need to reduce anterior spinal height as part of the operative procedure, if a satisfactory correction is to be achieved without tension lengthening the spinal cord.

The Surgical Management of Idiopathic Thoracic Scoliosis

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INTRODUCTION

In order to appreciate the necessary surgical strategy for idiopathic thoracic scoliosis it is essential to understand the three-dimensional nature of the deformity /9/. A postero-anterior (PA) radiograph of the thoracic spine of a patient with idiopathic scoliosis, Figure 1, shows two components of the deformity — lateral curvature and rotation. Rotation is always in the same direction in idiopathic scoliosis with the posterior elements directed towards the curve concavity and the anterior vertebral bodies towards the curve convexity. Thus it can be readily seen that a line joining the spinous processes from the top to the bottom of the curve describes a shorter distance than a line joining the centre of the vertebral bodies. Throughout the deformity, therefore, the back of the spine is shorter than the front and the whole deformity is lordotic /14, 27, 28/. The deformity of kypho-scoliosis (a kyphosis and a scoliosis are present at the same site) does not therefore exist. This point is of crucial significance in understanding how to correct the deformity in all three dimensions /10, 15, 16/.

Figure 1, being a PA view of the patient, demonstrates an oblique view of the deformity as the vertebrae are rotated and thus to obtain true planar views the beam or patient must be rotated accordingly. When a true lateral radiograph of the deformity is obtained the presence of the lordosis is confirmed, Figure 2. This lordosis is due to an alteration of vertebral shape in the sagittal plane, the intervertebral discs not contributing to the lordosis /9, 14/. With anterior vertebral height being greater than posterior height, with any



Fig. 1: Postero-anterior (PA) radiograph of a patient with idiopathic thoracic scoliosis. There is rotation such that the posterior elements are directed towards the curve concavity, while the bodies are directed towards the curve convexity. The posterior elements are shorter than the anterior elements and therefore the deformity is lordotic throughout.

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end-plate irregularity present more posteriorly, the deformity is precisely the opposite of the other common idiopathic spinal deformity of childhood and adolescence, Scheuermann's disease [14, 15]. Because the axis of spinal rotation passes in front of the kyphotically-shaped vertebrae of Scheuermann's disease,

that while any conservative treatment modality which causes spinal extension in Scheuermann's disease will lead to a true physiological correction of the deformity [5], with the idiopathic scoliotic deformity the lordosis would require flexion and this is when it is rotationally unstable [14, 15]. Thus the deformity of idiopathic

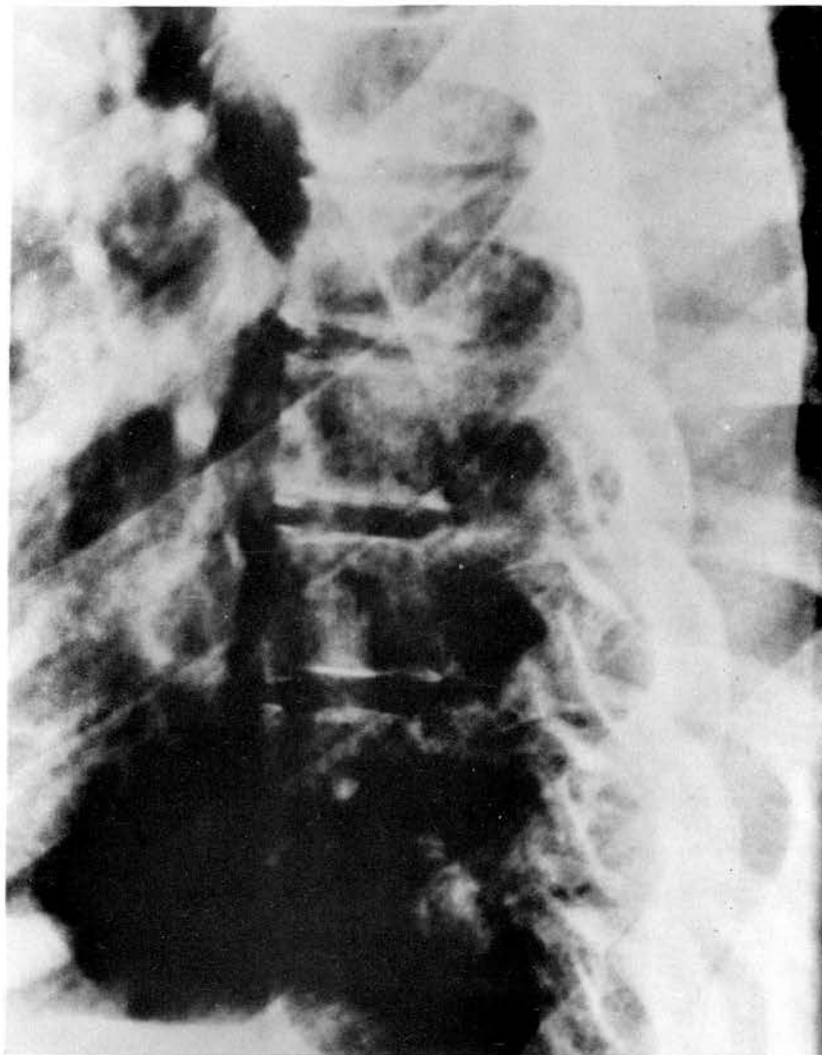


Fig. 2: True lateral radiograph of the apex of an idiopathic thoracic scoliosis. There is a lordosis at the curve apex and the vertebral bodies are wedged in the sagittal plane in the opposite direction to that encountered in Scheuermann's disease.

these are protected from rotation while, conversely, the lordotic vertebrae of idiopathic scoliosis lie anterior to this axis and are vulnerable to rotation [8], particularly on forward flexion (the rotational prominence is increased when the patient bends forward).

This biomechanical point is also crucial and indicates

scoliosis is not treatable conservatively [12] as long-term studies clearly suggest [7, 22].

So it is that the only method of effectively influencing the shape of the idiopathic scoliotic deformity is by surgical means.

MANAGEMENT PHILOSOPHY

There are two types of idiopathic deformity — early onset and late onset cases — for which the surgical management is quite different /10, 15, 16/. It is the early onset case which is associated with mortality, morbidity and the most severe deformities as there is that much more time for the deformity to progress /11, 12/. Therefore the primary aim of surgical inter-

vention is to prevent or minimise these organic consequences. In contradistinction the late onset adolescent idiopathic deformity, the one that is most commonly encountered, is purely a question of cosmesis, although the greater the deformity the more likely the social deprivation /26/ or psychological upset /3/. Moreover the late onset case presents as a result of the rotational prominence and it is this which requires correction. The standard method of attempting to achieve this



a

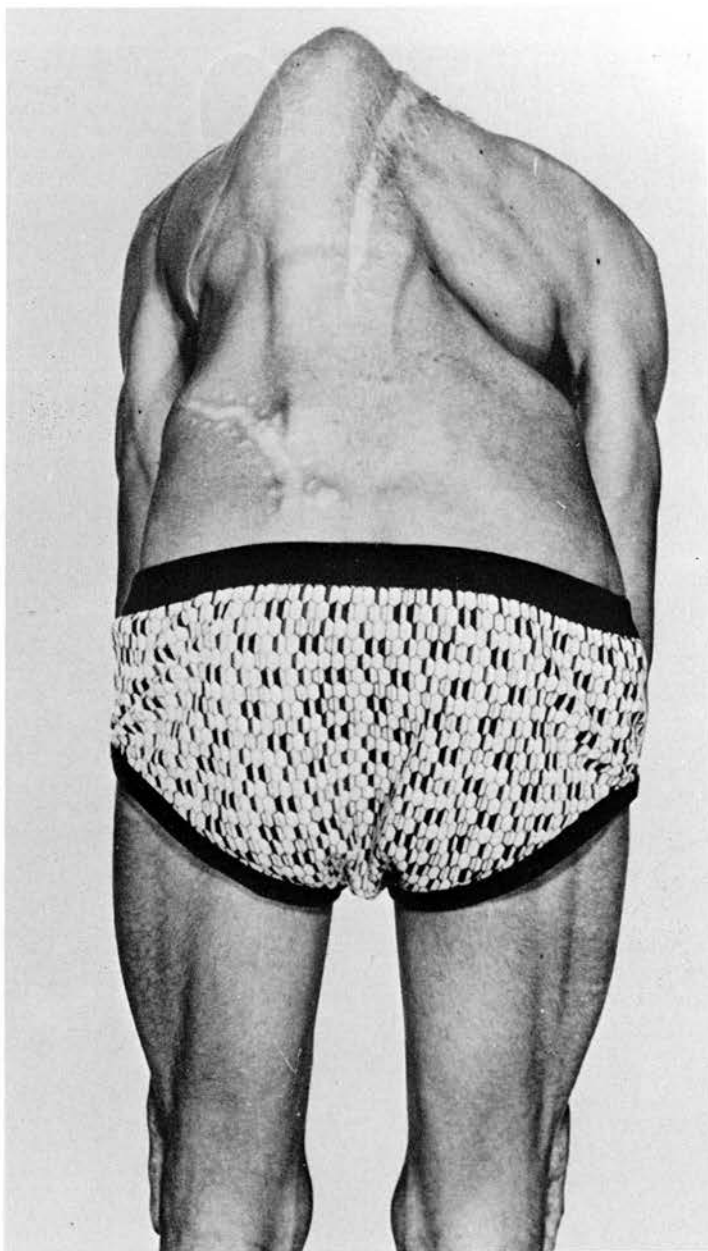
Fig. 3: The harmful effects of posterior fusion for an early onset progressive scoliosis:

(a) PA x-ray of the patient showing a mature posterior fusion which has rotated into the curve concavity thus tethering the spine;

is by the insertion of Harrington instrumentation /18, 19, 25/ but as this instrumentation is hooked into the spine above and below the deformity it does not have any significant effect on the rotational prominence /1, 4, 29/ and therefore the patient goes through a lot of surgery for little or no gain. The poor result from posterior instrumentation becomes all the more obvious with the bigger and more rigid curve and,

as traction of any kind has no beneficial effect on the scoliotic spine /13, 17/, the surgical attack requires to be more aggressive if a satisfactory correction is to be obtained /10, 16, 24/.

It is then customary to stabilize the "corrected position" by means of a spinal fusion which is traditionally performed posteriorly /18-20, 25/, to the great convenience of the surgeon. As can be seen from



b

Fig. 3: The harmful effects of posterior fusion for an early onset progressive scoliosis:

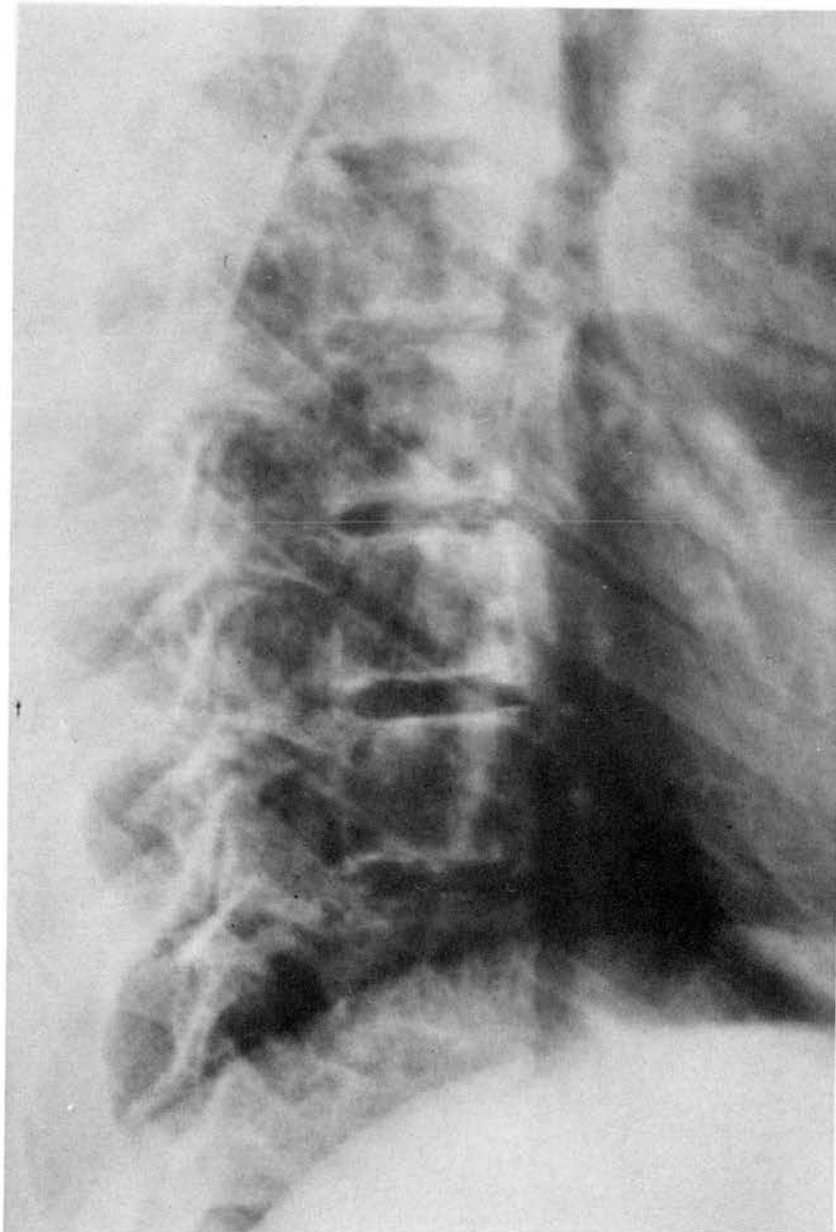
(b) clinical appearance of the patient showing the scars of posterior fusion and iliac crest bone graft removal. This is an end-stage situation as the deformity cannot get any worse.

the three-dimensional nature of the deformity /8, 9/, the back of the spine is already too short and thus it is quite possible by fusion to add insult to injury through posterior tethering /14, 15, 27/. For the late onset adolescent case with little progression potential a posterior spinal fusion may indeed halt subsequent deterioration but for the early onset progressive case quite the reverse frequently happens, Figure 3.

SIZE OF THE DEFORMITY

It is convenient to divide deformities into three categories as regards severity — mild (Cobb angle less than 60°), moderate (Cobb angle $60-90^\circ$) and severe (Cobb angle greater than 90° or previous posterior fusion). As the important deforming force in idiopathic scoliosis is growth then it is clear that the mild

Fig. 4: The technique of sublaminar wiring on the concave side of a Harrington distraction rod pre-bent to a normal thoracic kyphosis:



(a) lateral radiograph before surgery;

deformity is encountered most commonly with late onset scoliosis, and the severe deformity, often having had a previous posterior fusion, in the early onset group.

Mild Deformity

For this deformity the corrective instrumentation can be performed in one posterior approach. Dissat-

isfaction with the results of standard Harrington instrumentation [1, 4, 29] has led to the use of segmental spinal instrumentation [2, 6] (a combination of longitudinal rods and sublaminar wires bilaterally) or the combination of a Harrington distraction rod with sublaminar wiring on the concave side only. For reasons of safety the Harrington-Luque instrumentation approach is preferable to the bilateral segmental spinal instrumentation procedure. While these procedures

Fig. 4: The technique of sublaminar wiring on the concave side of a Harrington distraction rod pre-bent to a normal thoracic kyphosis:

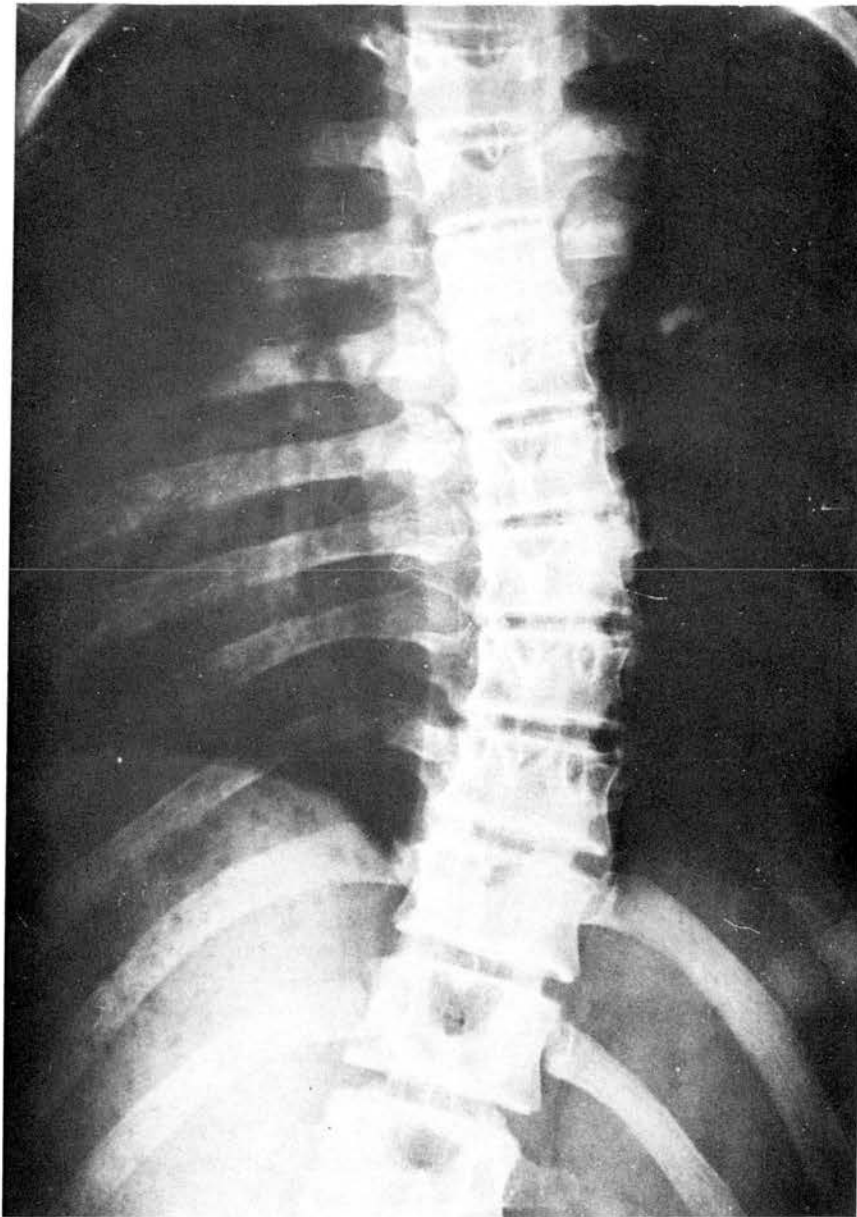


(b) lateral radiograph after surgery;

lead to an improved correction of the coronal plane component of the deformity the amount of apical rotation is either left unchanged or, in fact, increased. This is because the instrumentation is working on the wrong side of the axis of spinal rotation (similar to mini-compression systems at the curve apex). If concave sublaminar wires draw the spine to a straight concave rod then rotation cannot be corrected /10, 16/.

The necessary direction of pull of the concave wires must be directed more antero-posteriorly and this can only be achieved if the rod is bent into a kyphotic configuration in the sagittal plane, Figure 4 /10, 14-16/. Rotation of this rod is restricted by the use of a square ended lower hook-rod configuration. The three components of the deformity — rotation, lordosis and scoliosis — are all thereby corrected. For the late onset adolescent case with little or no progression potential

Fig. 4: The technique of sublaminar wiring on the concave side of a Harrington distraction rod pre-bent to a normal thoracic kyphosis:



(c) PA radiograph before surgery.

Fig. 4: The technique of sublaminar wiring on the concave side of a Harrington distraction rod pre-bent to a normal thoracic kyphosis:



(d) PA radiograph after surgery;

the superior correction obtained by this technique should be stabilised by a standard postero lateral spinal fusion, with the exception that the load bearing laminae on the concave wired side are not decorticated. Local flaps of bone are raised from the spinous processes, convex laminae, and the transverse processes bilaterally. On to these are bedded chips of homograft bone ob-

tained from the bone bank (it is not necessary to use the patient's own iliac crest bone which necessitates another scar, a longer operation, more blood loss, and a deal of post-operative discomfort).

If 'Harrington-Luque' instrumentation is used for the early onset progressive case then posterior fusion should be withheld, thus enabling the spine to continue to grow /10, 14-16/. If the thoracic kyphosis has been recreated by the instrumentation then the spine will have been brought into the correct relationship with its axis of rotation so that rotational progression is prevented. This "physiological approach" may indeed lead to further correction of the deformity with growth now that the mechanics are right.

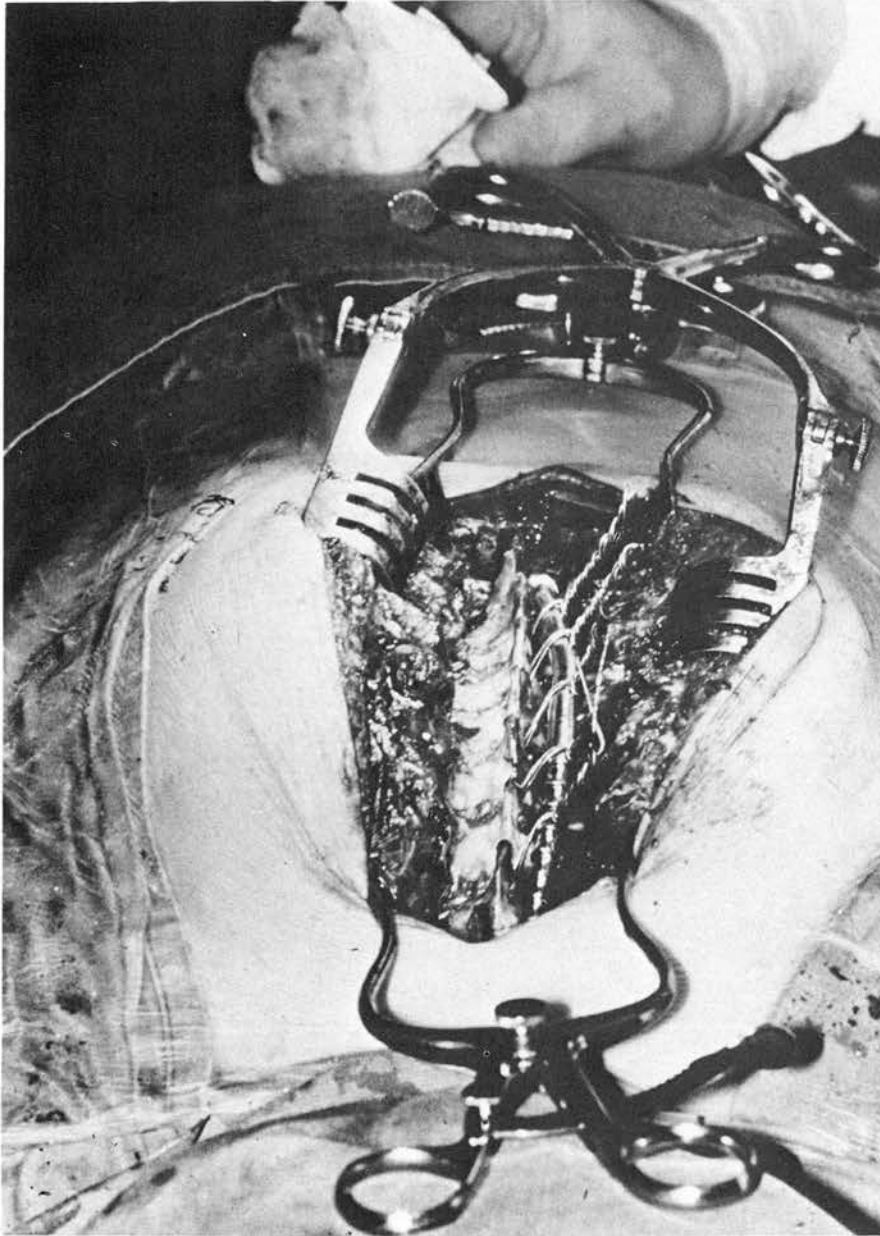
The Moderate Deformity

Here the deformity is both too great and too rigid to be handled by one posterior instrumentation procedure /10, 16/. As scoliosis and lordosis are positively correlated then the bigger the curve the more lordotic it is. Therefore, not only does rigidity prevent adequate correction but also there is a real danger of spinal lengthening, and thus spinal cord tension, occurring if the lordosis is brought back to the straight or even kyphotic configuration. In this situation the spine must be shortened, and, for the moderate deformity, this can be achieved by anterior discectomy in a preliminary first stage, Figure 5. The spine is approached through a standard thoracotomy so that there is no overhang preventing access to the spine within. Then as many discs as can be reached are removed and this is usually five or six. As these cases are typified by the front of the spine being too long then it is also physiologically appropriate to remove the vertebral end-plates as well as the discs so that anterior spinal growth is suppressed. In the second stage performed two to three weeks later the spine is instrumented using the same technique of concave sublaminar wiring to a kyphotic Harrington distraction rod. With first stage shortening there is good correction and the risks of a tension paraplegia are minimised /10, 16/.

The Severe Rigid Case

For this type of deformity, as with the severe rigid deformity of any aetiology, the only safe surgical strategy is to perform a closing wedge resection of the apex of the curve /24/ (Figure 6). This technique was pioneered by Leatherman in Louisville /23/ and again conforms to the important principle of not sub-

Fig. 4: The technique of sublaminar wiring on the concave side of a Harrington distraction rod pre-bent to a normal thoracic kyphosis:



(e) the spine being drawn to a pre-bent kyphotic rod (see colored slide).

jecting the spine to undue tension. The necessary sized wedge, based on the convex side, is removed in two stages, the anterior stage first with the second stage being performed posteriorly two or three weeks later. In the first stage, through a standard anterior thoracotomy, removing the rib above the apex of the deformity,

the vertebral body or bodies are resected at the curve apex in wedge fashion, more being taken from the convex side. The aim of the wedge removal is to come to a point on the concave side at the curve apex. As these deformities are usually significantly rotated lardo-scolioses the vertebral body plus the convex



Fig. 5: Anterior discectomy at multiple levels for the moderate idiopathic deformity which is beyond posterior instrumentation alone but not severe enough to warrant two-stage wedge resection.

pedicle and transverse process should be removed in the first stage. The concave pedicle and transverse process are not accessible from the front. Bone removal is performed using sharp osteotomes which take off successive slivers of bone until the spinal canal is breached. The remaining bone is removed using Cloward punches.

In the second stage wedge removal is completed posteriorly again coming to a point on the concave side. The pedicle and transverse process on the con-

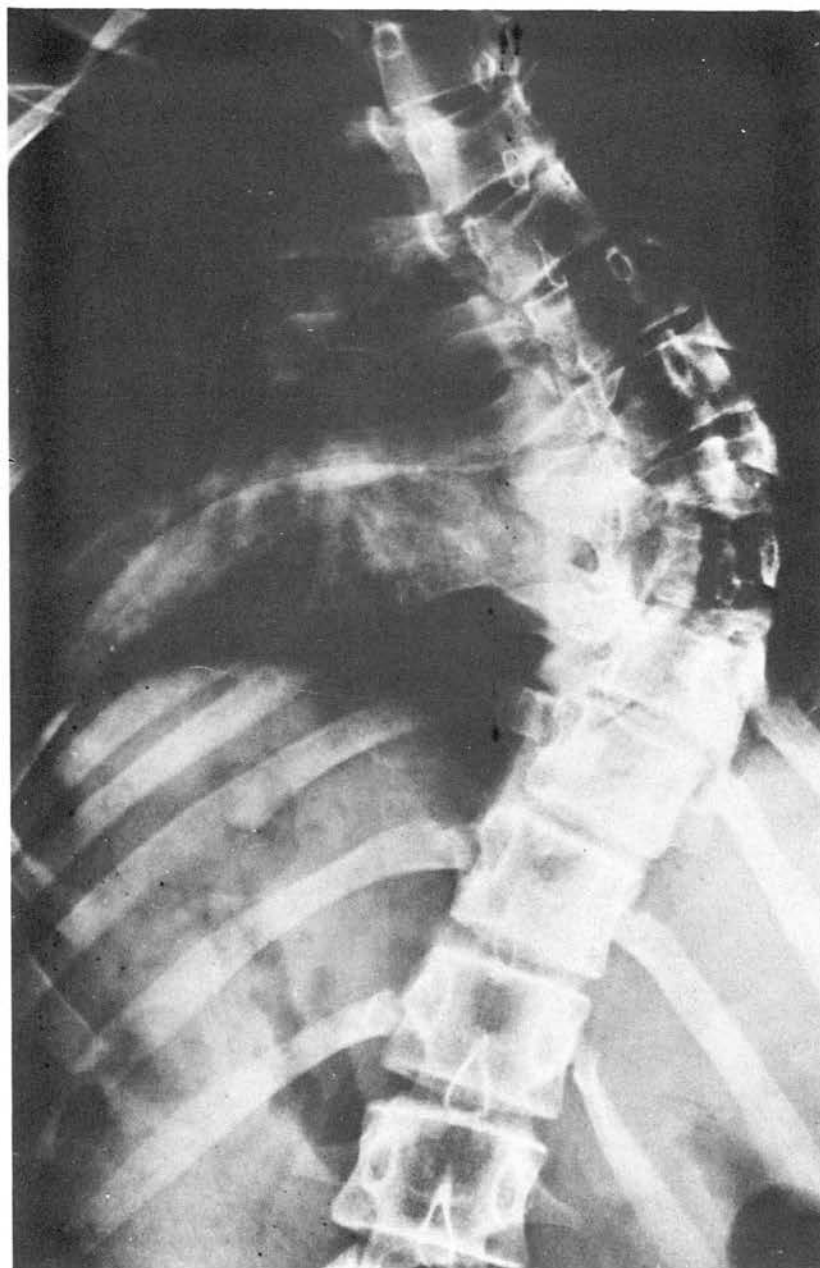
cave side are now removed and the wedge is ready for closure. This is performed by the insertion of a Harrington compression system on the convex side and, if possible, three compression hooks should bear down above the apex and three bear up from below. When the wedge is closed, and only then, is a Harrington distraction rod inserted on the concave side and this is more for stability than correction. If more of the back of the spine has been removed than the

Fig. 6: The two-stage wedge resection technique:**(a)** removal of the anterior component of the wedge (see colored slide)

front, then the front part of the wedge will close while the back is still open. As it is advisable to remove more bone posteriorly than anteriorly so that the spinal cord can be visualised when the wedge closes anteriorly there is often a small triangular gap at the back. This is bridged using local bone flaps and homograft bank bone if necessary.

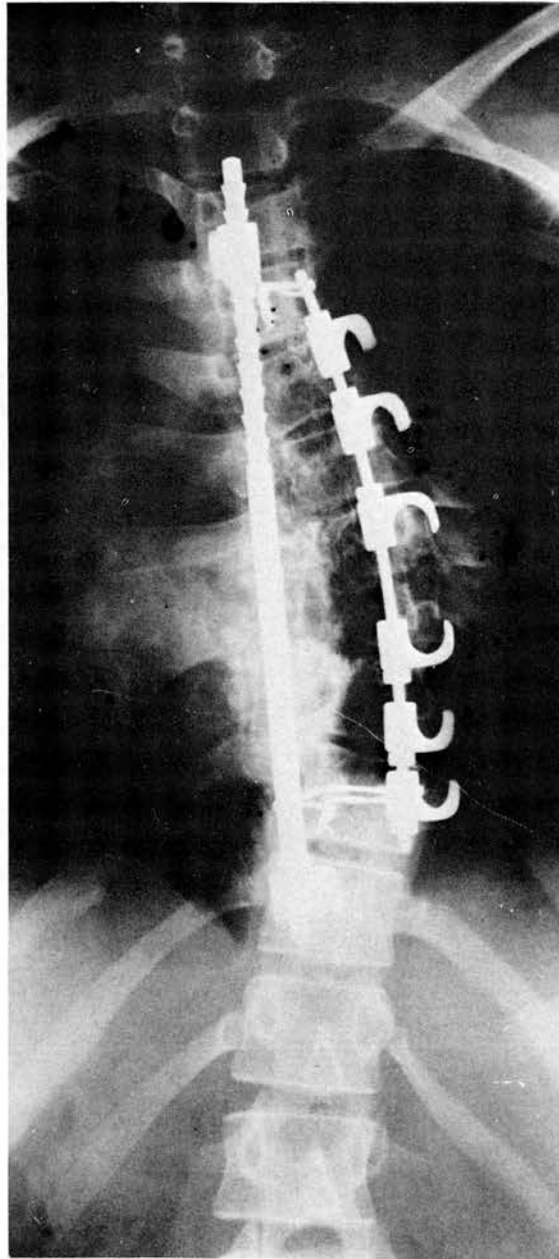
COSTOPLASTY

As the great majority of cases of idiopathic thoracic scoliosis which require operation are late onset cosmetic deformities then an optimal correction of the rib hump is essential. Furthermore, although earlier onset cases should be thought of primarily as organic problems,

Fig. 6: The two-stage wedge resection technique:**(b)** PA radiograph of a congenital thoracic curve before, and

there are still cosmetic considerations for the patient. If, after the above procedures, the rib hump remains unduly noticeable, and this is only the case with moderate or severe deformities, then division of the apical five or six ribs on the convex side should be performed, Figure 7. If these are osteotomised close to the trans-

verse process, and the lateral cut surface tucked under the medial, a very satisfactory reduction of the rotational prominence is achieved and this can be further improved by the application of a well fitting EDF (elongation-derotation-flexion) cast post-operatively. As a result we have not found it necessary to perform

Fig. 6: The two-stage wedge resection technique:

(c) after two-stage wedge resection. The wedge has been closed using a compression system on the convex side.

a more radical costoplasty as a separate stage later /21/.

POST-OPERATIVE MANAGEMENT

All patients are mobilised during the second post-

operative week after an EDF case is applied. For those whose correction has not involved rib division then the EDF cast is manufactured from polyurethane and is bi-valved with a zip-fastener down each side so that the patient can remove the cast for daily showering. This is a light-weight system and meets with con-

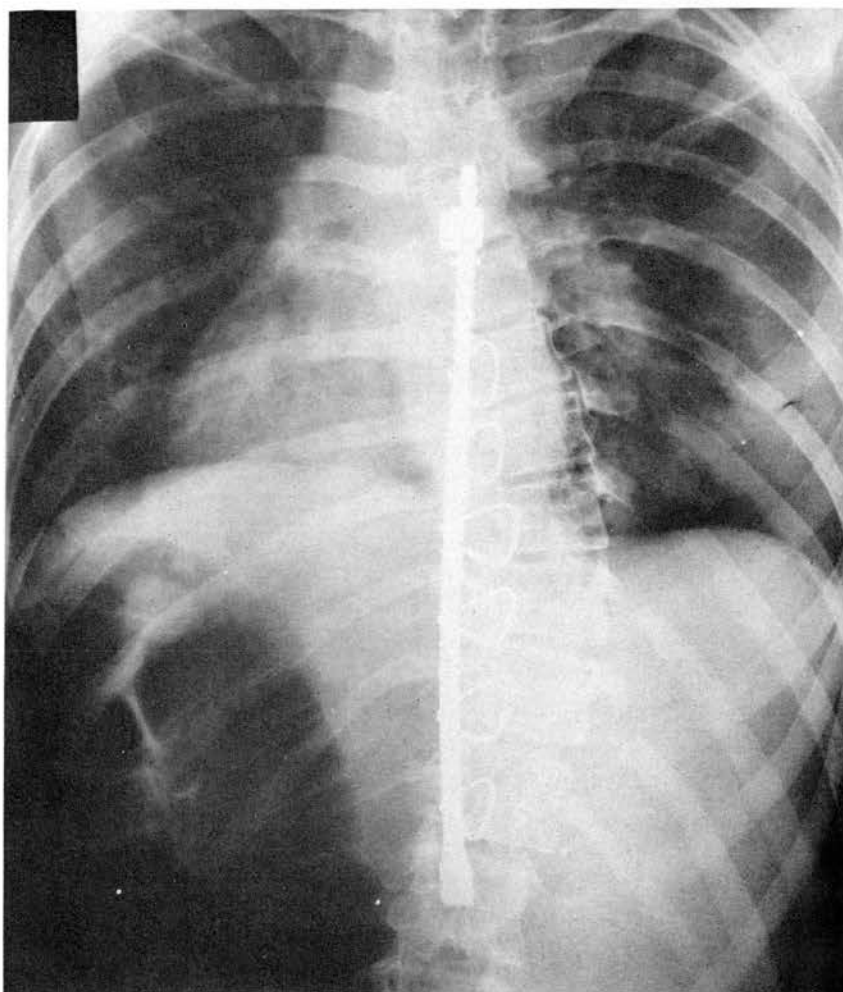


Fig. 7: Posterior instrumentation and fusion have been performed on this typical case of mild late onset idiopathic scoliosis, but the rotational prominence was still obvious on the operating table. Multiple rib osteotomies on the convex side have therefore been performed:

(a) PA radiograph of patient post-operatively,

siderable patient satisfaction, and thus, compliance. For those whose ribs have been divided the standard plaster of Paris EDF cast is prescribed as further sustained pressure over the convex ribs posteriorly is

required. All forms of immobilisation are removed at six months and the patient is free to resume any activity.

REFERENCES

1. Aaro S, Dahlborn M. The effect of Harrington instrumentation on the longitudinal axis rotation of the apical vertebra and on the spinal and rib-cage deformity in idiopathic scoliosis studied by computer tomography. *Spine* 1982; 7:456-462.
2. Allen BL. Segmental instrumentation of the spine: indications, results and complications. In: "Management of spinal deformities", edited by R.A. Dickson and D.S. Bradford, pp. 162-192. London, Butterworth, 1984.
3. Bengtsson G, Fallstrom K, Jansson B, Nachemson A. A psychological and psychiatric investigation of the adjustment of female scoliosis patients. *Acta Psychiat. Scandinavica* 1974; 50:50-59.
4. Benson DR, DeWald IL, Schultz Ab. Harrington rod distraction instrumentation. Its effect on vertebral rotation and thoracic compensation. *Clin. Orthop.* 1977; 125:40-44.
5. Bradford DS, Moe JH, Montalvo FJ, Winter RB. Scheuermann's kyphosis and roundback deformity. Results of Milwaukee Brace Treatment. *J. Bone and Joint Surg.* 1974; 56A:740-758.
6. Cardoso AM, Tajonar FA, Luque ED. Osteotomies de columna nuevos conceptos. *Anales de Ortopedia y Traumatologia* 1976; 12:105-113.
7. Carr WA, Moe JH, Winter RB, Lonstein JE. Treatment of idiopathic scoliosis in the Milwaukee brace. *J. Bone and Joint Surg.* 1980; 62A:599-612.
8. Deacon P, Archer IA, Dickson RA. The anatomy of spinal deformity - a biomechanical analysis. *Orthopedics* 1985; (in press).
9. Deacon P, Flood BM, Dickson RA. Idiopathic scoliosis in three dimensions. A radiographic and morphometric analysis. *J. Bone and Joint Surg.* 1984; 66b:509-512.
10. Dickson RA. Idiopathic scoliosis - foundation for a biological treatment. *Ann. Royal College of Surgeons* 1985; (in press).
11. Dickson RA. The conservative treatment of idiopathic scoliosis. *J. Bone and Joint Surg.* 1985; (in press).
12. Dickson RA, Archer IA. Scoliosis in the community. In: "Management of Spinal Deformities", edited by R.A. Dickson and D.S. Bradford, pp. 77-100. London, Butterworth, 1984.
13. Dickson RA, Leatherman KD. Cotrel traction, exercises, casting in the treatment of idiopathic scoliosis. *Acta Orthop. Scandinavica* 1978; 49:46-48.
14. Dickson RA, Lawton JO, Archer IA, Butt WP. The pathogenesis of idiopathic scoliosis - biplanar spinal asymmetry. *J. Bone and Joint Surg.* 1984; 66B:8-15.
15. Dickson RA, Lawton JO, Butt WP. The pathogenesis of idiopathic scoliosis. In: "Management of Spinal Deformities", edited by R.A. Dickson and D.S. Bradford, pp. 1-37. London, Butterworth, 1984.
16. Dickson RA, Lawton JO, Deacon P, Archer IA, Flood BM. The pathogenesis of idiopathic scoliosis. In: "Pro-

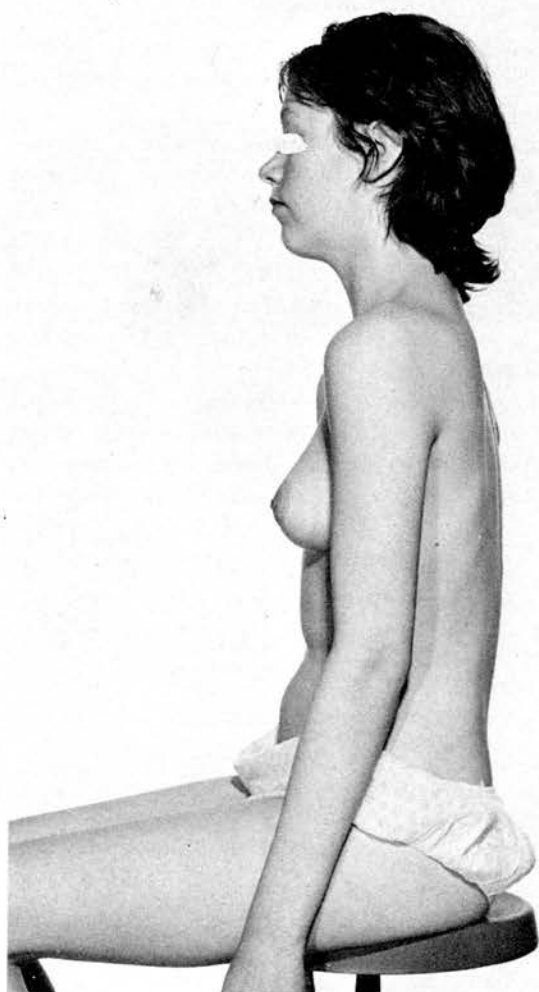


Fig. 7: Posterior instrumentation and fusion have been performed on this typical case of mild late onset idiopathic scoliosis, but the rotational prominence was still obvious on the operating table. Multiple rib osteotomies on the convex side have therefore been performed:

(b) side view of patient at follow-up showing no discernible rotational prominence.

- ceedings of the 7th Philip Zorab Symposium 1983 – Prevention”, edited by J. Warner and M. Mehta. New York, Praeger, 1985 (in press).
17. Edgar MA, Chapman RH, Glasgow MMS. Pre-operative correction in adolescent idiopathic scoliosis. *J. Bone and Joint Surg.* 1982; 64B:530-535.
 18. Goldstein LA. Surgical management of scoliosis. *J. Bone and Joint Surg.* 1966; 48A:167-196.
 19. Harrington PR. Surgical instrumentation for management of scoliosis. *J. Bone and Joint Surg.* 1960; 42A:1448.
 20. Hibbs RA. An operation for progressive spinal deformities. *New York Med. J.* 1911; 93:1013-1016.
 21. Houghton GR. Cosmetic surgery for scoliosis. In: “Management of Spinal Deformities”, edited by R.A. Dickson and D.S. Bradford, pp. 237-251. London, Butterworth, 1984.
 22. Kaiser RP, Shufflebarger HL. The Milwaukee Brace in idiopathic scoliosis. Evaluation of 123 completed cases. *Clin. Orthop.* 1976; 118:19-24.
 27. Roaf R. The basic anatomy of scoliosis. *J. Bone and Joint Surg.* 1966; 48B:786-792.
 28. Somerville EW. Rotational Lordosis: the development of the single curve. *J. Bone and Joint Surg.* 1952; 34B: 421-427.
 29. Thulbourne T, Gillespie R. The rib hump in idiopathic scoliosis. Measurement analysis and response to treatment. *J. Bone and Joint Surg.* 1976; 58B:64-71.
 23. Leatherman KD. The management of rigid spinal curves. *Clin. Orthop.* 1973; 93:215-224.
 24. Leatherman KD, Dickson RA. Two-stage corrective surgery for congenital deformities of the spine. *J. Bone and Joint Surg.* 1979; 61B:324-328.
 25. Moe JH. Methods of correction and surgical techniques in scoliosis. *Orthop. Clin. North America* 1972; 3:17-48.
 26. Nachemson A. A long term follow up study of non-treated scoliosis. *Acta Orthop. Scandinavica* 1968; 39:466-476.
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SPINAL DEFORMITIES IN CHILDREN.

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Surgery; 1: 514-524, 1985.

The aetiology and clinical features of idiopathic and other spinal deformities was describe in relation to pathomechanical principles and the necessary surgical treatment described.

Spinal deformities in children

When the spine is viewed anteriorly or posteriorly it should be straight and any curvature in this, the coronal plane, is termed a 'scoliosis'. At the end of the first year, when a child begins to stand and walk, normal lateral profile (sagittal plane) curvatures become obvious. These are convex anteriorly in the cervical and lumbar regions and are called 'lordoses', while the thoracic region remains convex posteriorly; this is a 'kyphosis'. If these sagittal curvatures are exaggerated, or if they occur in a direction not normally encountered at that site, they assume clinical significance.

Abnormalities of scoliosis, kyphosis, or lordosis may exist in combination; they are referred to as structural or non-structural according to the nature and location of the underlying problem. Inequality in leg length which tilts the pelvis, or painful paravertebral muscle spasm accompanying a prolapsed disc, produce their non-structural scolioses as a secondary phenomenon. These are mild and non-progressive conditions which usually resolve when the underlying problem is dealt with (e.g. shoe raise or disc removal).

Structural deformities are much more serious; they may progress with growth and are caused by deforming forces either in the spine or in its supporting structures. Thus, a congenital failure of vertebrae to segment on one side produces a structural spinal deformity, and, because growth is also impeded on the unsegmented side the deformity will progress with time. Failure of the paravertebral muscles to support the growing spine in a normal and symmetrical fashion also favours progression of a spinal deformity; thus, poliomyelitis and cerebral palsy are commonly associated conditions. The child with spina bifida may have both congenital bony and paralytic reasons for developing the deformity. An aetiological classification,

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therefore, has some merit in bringing together the various conditions in which spinal deformities are prevalent.

Pathomechanics

The pathomechanics of spinal deformity are crucial in understanding the clinical behaviour and treatment requirements. Pure scolioses (coronal plane curves) are uncommon, unimportant, and usually non-structural and non-progressive. The feature distinguishing structural scolioses is rotation. Figure 1 is a posteroanterior (PA) radiograph of a patient with idiopathic thoracic scoliosis; in addition to the lateral curvature of the spine, there is vertebral rotation with the vertebral bodies anteriorly being rotated into the curve convexity and the posterior elements into the curve concavity. This direction of rotation is constant regardless of the type or site of the curve.

It is necessary to think of the spine in three dimensions. If the posterior elements are towards the concavity and the bodies are toward the convexity, a line connecting the former will be shorter than a line connecting the latter, and thus the back of the spine is shorter than the front throughout the curve. The deformity of structural scoliosis is, therefore, lordotic throughout - this is somewhat unexpected as in the thoracic

region it is normal to have a kyphosis which has, therefore, been reversed in the development of the deformity. The axis of spinal rotation (Figure 2) which normally passes in front of kyphoses and behind lordoses is now behind the thoracic spine. This accordingly comes under compression and must rotate to the side in order to be accommodated, in so doing producing the scoliosis as a secondary phenomenon. There is already a lordosis in the lumbar region and, therefore, if a bony scoliosis is superimposed, or the paravertebral 'guy ropes' fail asymmetrically, a lordoscoliosis is similarly produced.

Kyphotic conditions (e.g. Scheuermann's disease or the kyphosis of infection or tumour) lie behind this axis and are thereby protected from rotation. Thus, for practical purposes there are really only two types of deformity, lordosis and kyphosis, the former being rotationally unstable and swinging out to the side to provide the coronal plane component to the three dimensional deformity, while the latter remains in the sagittal plane. As a corollary, the deformity of kyphoscoliosis (a kyphosis and scoliosis at the same site) cannot and does not exist!

Radiographic evaluation

Because the deformity of structural scoliosis encompasses both lateral curvature and rotation, Figure 1, although a PA view of the patient, necessarily becomes an oblique view of the deformity, the degree of obliquity depending on the degree of rotation. Despite this obvious problem, the standard method for determining curve size radiographically is by drawing lines along the maximally tilted vertebrae at the top and bottom of the curve and then measuring the angle which these lines subtend, on a PA view of the patient (and not the deformity). This is referred to as the Cobb angle. Though accurate for uniplanar kyphoses, the

Aetiological classification

Cause	Subgroups	L	CL	K
• Idiopathic	Idiopathic scoliosis Idiopathic kyphosis (Scheuermann's disease)	+		+
• Congenital	Bone anomalies Cord anomalies Bone and cord anomalies	+		+
• Neuromuscular	Poliomyelitis Cerebral palsy True neuromuscular disorders (spinal muscle atrophy, peripheral neuropathies; Friedreich's ataxia; arthrogryposis; the muscle dystrophies)	+	+	
• Neurofibromatosis	Mild disease Dystrophic disease	+		+
• Heritable disorders of connective tissue, mucopolysaccharidoses, bone dysplasias, metabolic, endocrine	Osteogenesis imperfecta Other heritable disorders Mucopolysaccharidoses Bone dysplasias Metabolic/endocrine	+		+
• Trauma	Vertebral Injury Laminectomy Extravertebral Chest operations		+	+
• Infection	Osteomyelitis Tuberculosis			+
• Tumours	Intradural Extradural ± radiation	+		+

L - lordoscoliosis; CL - collapsing lordoscoliosis with pelvic obliquity;
K - kyphosis.

Cobb angle, though much used, is of no real value in the measurement of rotated lordoscolioses.

Figure 3 explains this error and why the spurious appearance of a kyphoscoliosis is produced. As the PA view of the patient is an oblique view of the deformity, the same will be the case with a lateral view of the patient, and what is misinterpreted as a kyphosis is nothing more than the scoliosis itself. Indeed, if there are 45° of apical rotation, then PA and lateral views of the patient will produce identical oblique projections of the same scoliosis (although referred to as, say 50° of scoliosis and 50° of kyphosis respectively). To prevent Pythagoras 'turning in his grave', either patient or X-ray beam must be turned according to the amount of apical rotation so that the

deformity becomes maximal. The plane at 90° from this is where a true lateral projection of the deformity is obtained. Now the spine is straightest but the apical vertebrae are always lordotic. This error bedevils practically all reports on the natural course of spinal deformities and their response to treatment.

Idiopathic deformities

Scoliosis

Apart from the scoliosis, the children are essentially normal and, in particular, there is no evidence of any congenital spinal malformation or paralysis. There are two types of idiopathic scoliosis - early and late onset (before and after 5 years of age respectively). The early onset variety

is more common in boys, the thoracic site is most usually affected, and curves tend to be convex to the left. Moulding features are present elsewhere; these include plagiocephaly, torticollis, plagio pelvis and hip adduction. In more than 90% of cases the babies are of normal birth weight, are active and healthy and the condition resolves spontaneously. Less than 10% are progressive, but these, if untreated, produce both severe deformity and cardiopulmonary compromise with early death. Babies who are hypotonic with a low birth weight are vulnerable to progression.

Late onset idiopathic scoliosis is solely a spinal deformity, though the more growth remaining, the more significant the deformity can be, sometimes with social and psychological implications. Screening teenagers has demonstrated that 15% of schoolchildren appear to have some degree of scoliosis; 2% have curves measuring 10° or more and 0.2% curves measuring 20° or more. Thus, minor degrees of idiopathic scoliosis, usually non-progressive, are common; boys and girls are equally affected with the lumbar site being the most usual. Curves of clinical significance are found more often in girls, and right thoracic curves are most likely to progress.

These are all lordoscolioses and true lateral radiographs demonstrate lordotic vertebral body wedging at the apex of the curve, disc height not being a contributory factor. There is a spectrum of vertebral lateral profiles in the thoracic region; at one end are children with very flat lateral profiles, some of whom are truly lordotic and thus in danger of rotating, while at the other are those with such an excessive thoracic kyphosis that they may be categorized as suffering from Scheuermann's disease. (This affects the same area of the thoracic spine (T8-9), is also familial and has a similar prevalence.)

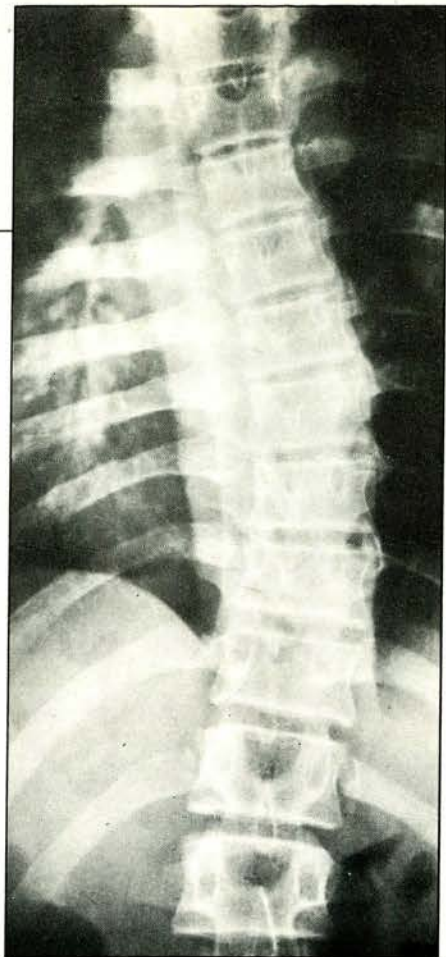
Kyphosis (Scheuermann's disease)

There are two types of Scheuermann's disease - type 1, an increased thoracic kyphosis, apical at the T8-9 level, and type 2, a kyphotically shaped thoracolumbar or upper lumbar vertebra which results from stress and is referred to as 'apprentices' spine'. Type 1 disease is the opposite condition to idiopathic scoliosis (Figure 4). Lateral radiography demonstrates the wedging which must be 5° of kyphosis per vertebral body extending over three vertebrae; the deformity tends to be rigid. Although the condition is equally common in boys and girls, more girls attend spinal deformity clinics, presumably because they are more concerned about their physical appearance.

The average age of presentation (15 years old) is approximately 2 years later than that of late onset idiopathic scoliosis; this can be attributed to the regaining of the normal thoracic kyphosis which occurs towards the end of adolescence, whereas early in adolescence the lateral profile of children is flattest. Pain may be a feature of Scheuermann's disease, either locally (probably caused by accelerated vertebral growth during adolescence) or in the lower lumbar region (where there is an increased prevalence of spondylosis secondary to the compensatory increased lumbar lordosis).

Congenital bony deformities

Occasionally, a solitary failure of formation (a single hemivertebra) produces a pure scoliosis and this tends to be non-progressive and of little consequence. More importantly, multiple failures of formation or, more usually, segmentation on the same side of the spine give rise to severe and progressive deformities. As with idiopathic scoliosis, the deformity is a lordoscoliosis (Figure 5).

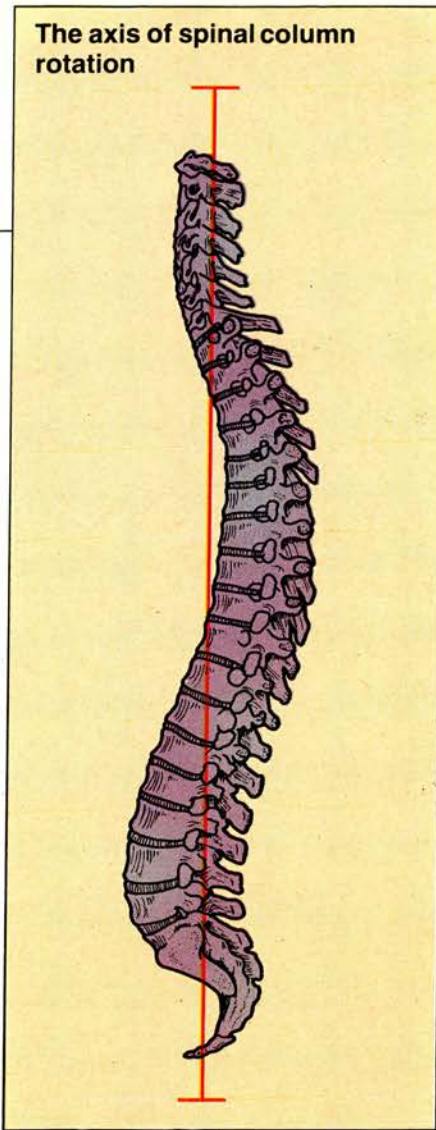


1 A PA radiograph of a patient with idiopathic thoracic scoliosis. There is a lateral curvature of the spine with rotation, such that the posterior elements are directed towards the curve concavity and thus pursue a shorter distance through the curve than the vertebral bodies, confirming that the deformity is lordotic throughout.

This is because the posterior elements are also involved in the failure process and thus a true lordosis is present. If the developmental failure is situated anteriorly, an angular kyphosis is produced and the spinal cord may be stretched causing paralysis.

In these children, the extraskeletal aspects must be carefully evaluated; more than 50%, for example, have an abnormality on an intravenous urogram (IVU). Moreover, 10% of such patients have evidence of spinal dysraphism - either a diastematomyelia (a midline bony or fibrous peg traversing the cord) or a tethered filum terminale; these may require resection or release before treatment is contemplated.

With such bony deformities there is a range of progression - while the



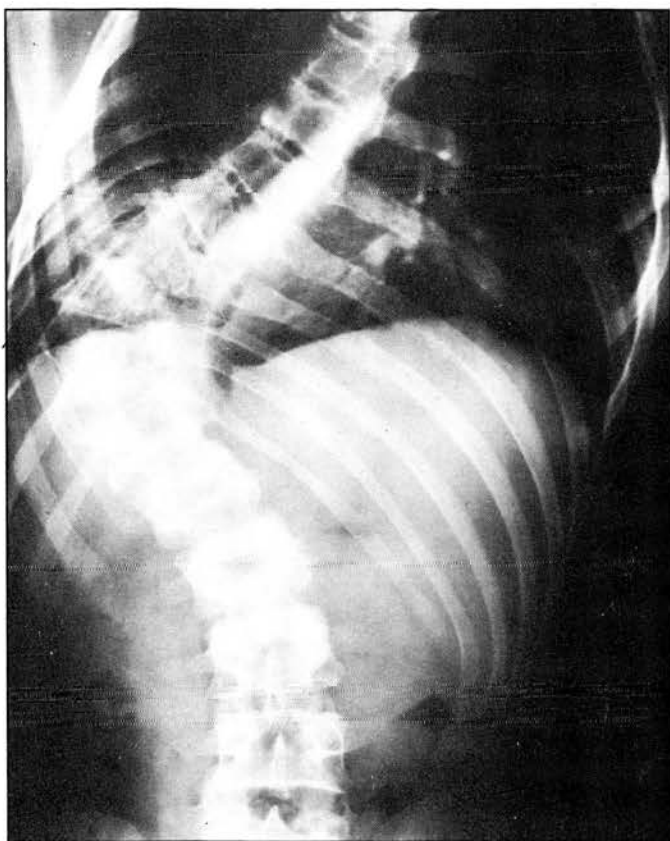
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most severe present very early, some babies have a mild failure in development which is not precipitated until the adolescent growth spurt. The diagnosis may not be obvious clinically but radiography will demonstrate the congenital bony anomaly.

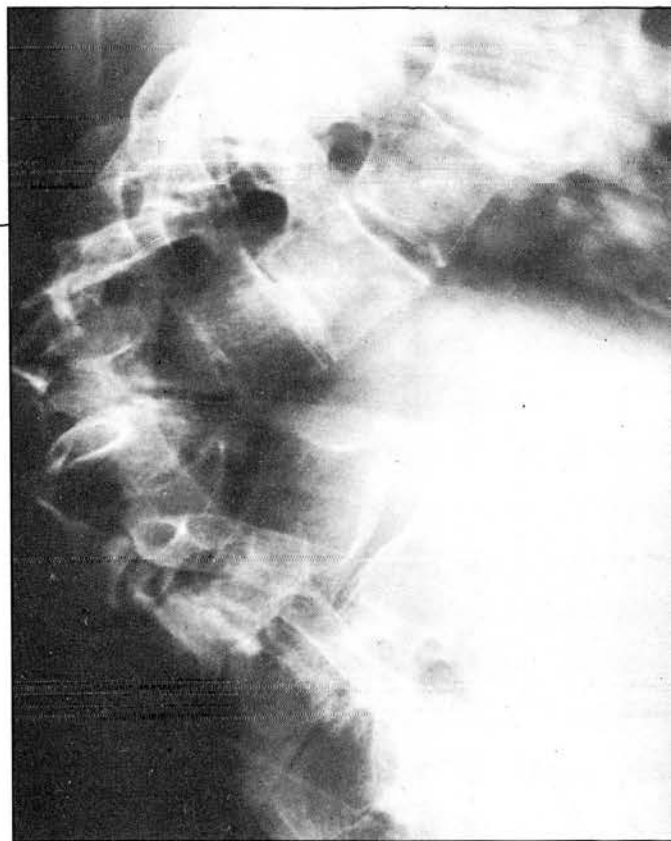
Cutaneous stigmas (e.g. a tuft of hair, lipoma, haemangioma, naevus, dimple, pit, sinus or fistula in the midline, and usually in the lumbar region) should raise suspicions of a congenital deformity. Congenital bony deformity is more rigid than the idiopathic counterpart and thus side bending does not tend to diminish its size.

Congenital cord deformities

Spina bifida syndrome: paralytic failure of the paravertebral muscles



7 The angular dystrophic rotated lordoscoliosis of Von Recklinghausen's disease.



8 Lateral radiograph of the spine showing the kyphosis produced by spinal infection.

occulta and thus the cutaneous stigmata should be sought. This is important if distraction instrumentation is to be used, lest a neurological catastrophe be precipitated by cord tethering. The integument is also inspected for the *café au lait* spots suggestive of neurofibromatosis. The flexibility of the deformity can be assessed in the standing position by lateral spinal flexion for scoliosis and by spinal extension for kyphoses. The patient is then examined in the supine position and while straight leg raising is not reduced in lordoscolioses, it is usually reduced by approximately 25% in Scheuermann's disease because of hamstring tightness. A neurological examination of the lower extremities should follow. Standing height, sitting height and weight are measured and the degree of secondary sexual development recorded. This is essential in estimating growth velocity and maturation and, thereby, progression potential.

Other deformities: there are usually obvious signs on inspection if the deformity is not idiopathic. Disproportionate short stature (dwarfism)

occurs with the mucopolysaccharidoses and skeletal dysplasias, while ligament laxity or joint contracture are associated with heritable disorders of connective tissue. The spina bifida child is usually recognized by the lower spinal scar, obvious paralysis, hydrocephalus or strabismus; many of the neuromuscular conditions will have already been diagnosed, the spinal deformity being a relatively late manifestation. Nevertheless, the establishment of the correct diagnosis is critical so that the nature and prognosis of the underlying condition is known. Careful neurological examination, muscle biopsy and muscle enzyme estimation may all be required.

Investigations

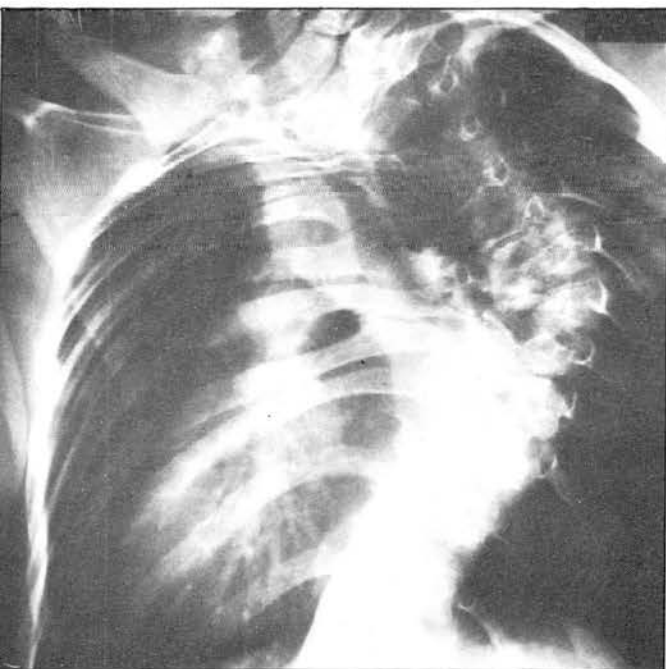
Radiography: it is traditional to take a PA radiograph of the patient's spine to exclude an obvious bony cause for the deformity. True planar views will be required to measure the size of the deformity.

In order to unravel the precise diagnosis of less common conditions, additional radiographs are necessary (hips, shoulders and hands for the

dysplasias; wrists and knees for rickets etc). In addition, those heritable disorders of connective tissue and mucopolysaccharidoses that are based upon known biochemical defects should have these established.

Radiographs of the hand and wrist should be taken routinely to establish bone age, as the chronological age of the patient is a much less precise determinant of true biological age. At general skeletal maturity (ages 14 years in girls and 17 in boys) the spine is still growing. The vertebral epiphyses do not fuse until the middle of the third decade which thus provides a reason why most curves progress until then. This must be borne in mind when treatment is planned. The presenting rotational deformity should be recorded photographically and surface shape topographically so that change can be quantified.

Myelography is indicated because of the high prevalence of spinal dysraphism in congenital spine deformities (Figure 10). This can be conveniently combined with computed tomography (CT). In other conditions (e.g. neurofibromatosis)



5 Congenital thoracic scoliosis caused by failure of segmentation; a lordoscoliosis.



6 PA radiograph of a collapsing lordoscoliosis associated with spina bifida.

Neurofibromatosis

In Von Recklinghausen's disease all components of the musculoskeletal system, including the vertebrae, are affected by the pathological process. As a result, 30% of children with neurofibromatosis have a lordoscoliosis measuring 20° or more. There is a spectrum of curves from the mild idiopathic-type at the one end, to the short, sharp angular dystrophic curve with a great deal of rotation, at the other (Figure 7). This correlates well with the degree of involvement with Von Recklinghausen's disease. A kyphosis is also common and varies from the mild idiopathic to the angular dystrophic.

Mesenchymal disorders

Spinal deformity may be associated with connective tissue disorders, mucopolysaccharidoses, bone dysplasias, skeletal dysplasias, and with metabolic and with endocrine disturbances.

In osteogenesis imperfecta there is a spectrum of deformity similar to that encountered in neurofibromatosis, while Marfan's syndrome, homocystinuria and Ehlers-Danlos syndrome are associated with the idiopathic type of curvature. The mucopolysaccharidoses are associ-

ated with platyspondyly (abnormally flattened vertebrae) often with a thoracolumbar anterior wedged vertebra giving rise to an angular kyphosis. The skeletal dysplasias also have a tendency toward a thoracolumbar kyphosis with a wedged vertebra, but some (e.g. diastrophic dwarfism) are associated with the dystrophic type of deformity encountered in Von Recklinghausen's disease.

In rickets there is a lordoscoliosis which is indistinguishable from the idiopathic type.

Other deformities

Vertebral trauma: injury or laminectomy can be the cause of a progressive kyphosis, while extravertebral trauma (e.g. after rib resection) produces an idiopathic type of lordoscoliosis.

Infection: spinal deformity associated with infection involves the intervertebral disc and its contiguous vertebral margins so causing a kyphosis (Figure 8). If the infection is severe, or is associated with abscess formation, neurological signs may develop.

Tumours: a local kyphosis is usually produced by extradural vertebral tumours, whether primary or second-

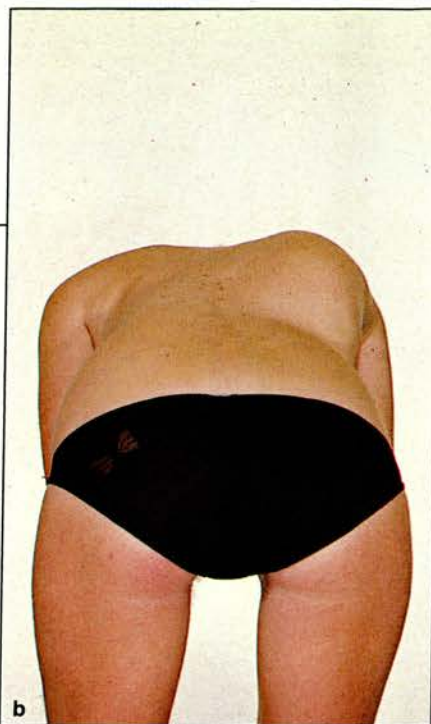
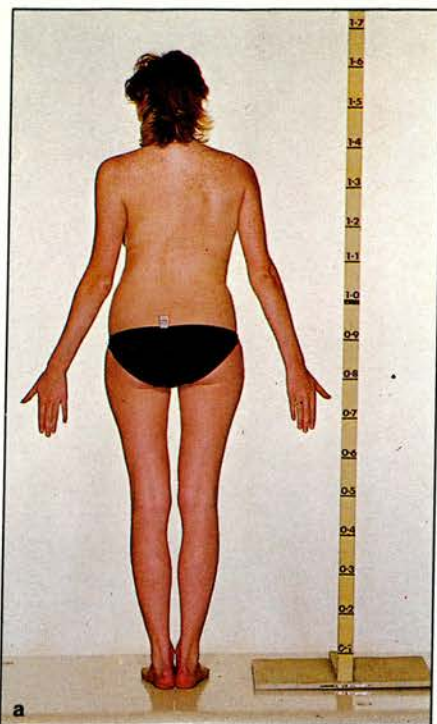
dary. The uncommon, but painful osteoblastoma or osteoid osteoma, occurring in the posterolateral elements, can produce a non-structural curve through a painful muscle spasm. An intradural tumour can present with an idiopathic type of curvature but the mechanism is unclear.

Diagnosis

Physical examination

Idiopathic deformities: most deformities encountered are idiopathic. The patient is examined naked, first in the erect position. The type and location of the deformity is noted (e.g. right thoracic or left lumbar scoliosis, or lower thoracic kyphosis). The patient is then asked to bend forward. The lordoscoliosis will rotate because it is under compression, producing a marked increase in the rotational prominence associated with the curve (Figure 9). With Scheuermann's disease, the forward flexion position exaggerates the lower thoracic kyphosis and may produce a loin hump below the kyphosis - in half of all patients with Scheuermann's disease there is also a mild idiopathic scoliosis below the area of kyphosis caused by rotation of the compensatory lordosis.

Approximately 10% of the 'normal' population have a spina bifida



9 A patient with idiopathic thoracic scoliosis in the erect position (a) and on forward flexion (b) which causes the rotational prominence to be maximized.

corrected conservatively, then the lordosis must be reversed and this implies spinal flexion. Unfortunately, it is precisely flexion which causes the rotational instability and this is why the forward bending position is an integral part of the clinical examination. Lordoscolioses cannot, therefore, be treated by conservative means. As most mild curves do not, in fact, progress, major surgery cannot be routinely prescribed. A period of observation is necessary to see if the curve deteriorates towards unacceptability.

What is unacceptability? Old-fashioned dogma would say that a Cobb angle approaching 60° was unacceptable because of the likelihood of cardiopulmonary complications, a fear which is now known to be unjustified for the late onset case. The deformity is, therefore, the pertinent matter and it is the patient and family who must decide what is acceptable and what is not. The surgical aim will then be to restore acceptability and preserve it. The standard method is by the insertion of Harrington instrumentation which takes up the natural elasticity of the curve. Curve size and flexibility are crucial; for flexible curves a correction of the coronal plane component by 50% can be achieved, but as curve size increases so does rigidity, and little or no correction is possible. Moreover, because the distraction forces are exerted at the top and bottom of the curve, there is no significant alteration in the rotational prominence, even with mild and flexible deformities.

With the advent of Luque's segmental wiring (passing wires under the laminae of each vertebra in the curve and thereby drawing the vertebrae to a longitudinal rod or rods) it is possible to obtain an improved correction in all three planes. This system was first introduced to deal more effectively with collapsing

Management

Management depends upon four principal factors:

- the nature of the deformity (type, size and rigidity)
- progression potential
- the presence or threat of paralysis
- the underlying condition.

The nature of the deformity

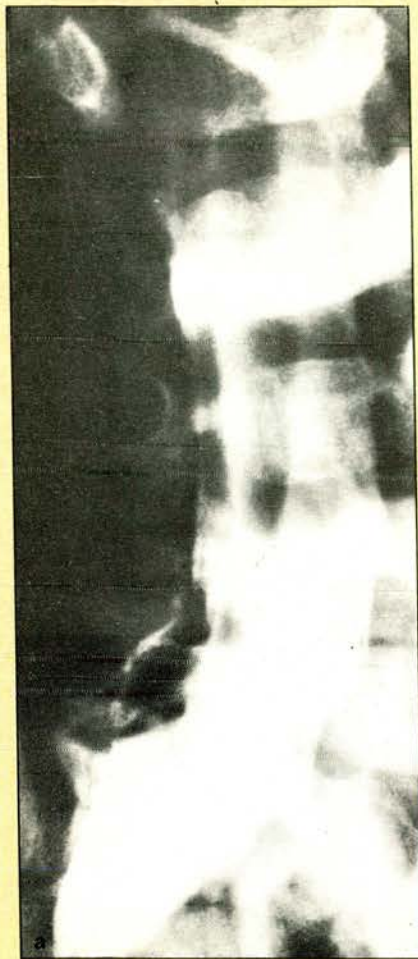
First, it is necessary to grasp the essentials of the management of idiopathic deformities so that variations on this theme in dealing with more complex deformities can be more readily understood. Late onset idiopathic scoliosis is a question solely of deformity and the first question to be asked is 'Is the deformity acceptable?' If so, then preservation of acceptability is the aim. Unfortunately, no conservative treatment is known which alters the natural course of this condition. The Milwaukee brace, for example, was designed to support the poliomyelitis scoliosis after fusion and not for the preoperative treatment of any form of scoliosis.

Appreciation of the three dimensional problem clearly indicates that a brace or cast would not be effective. If, for example, the deformity of idiopathic thoracic scoliosis was to be

and the skeletal dysplasias) and where there has been previous surgery, the demonstration of a normal spinal canal by CT myelography is an important safety precaution.

Intravenous urography: because urological anomalies are commonly associated with congenital spinal deformities an IVU is essential.

Cardiopulmonary assessment: only early onset deformities are associated with cardiopulmonary embarrassment of such severity that heart and lung failure are the cause of early mortality. Accordingly, a cardiopulmonary assessment is important. In such patients, spirometric lung function values may be only a small percentage of normal. With late onset scoliosis there is no significant cardiopulmonary compromise, though with bigger curves spirometric testing often reveals a mild restrictive defect because of the associated lack of chest wall flexibility. Such assessment is, however, critical as it would be totally inappropriate to offer corrective surgery to a patient whose cardiopulmonary system is already suffering from irreversible damage, or to offer a chance of preventing cardiopulmonary compromise in the future when no such danger exists.



(a) PA myelographic appearance of a patient with a diastematomyelia which splits the spinal cord.
(b) Photograph of the same patient showing shortening and attenuation of the right lower extremity.

the convex side and a stabilizing distraction rod is inserted on the concave side (Figure 12).

Should the deformity lie in the lumbar region, the same strategy is employed. A very poor posture is obtained if a straight or kyphotic rod is used in this situation and, therefore, a lordotic rod will be necessary to preserve the lumbar lordosis – optimal derotation may, therefore, have to be sacrificed with lumbar curves.

This same treatment strategy is used for all other idiopathic types of curves (e.g. Marfan's syndrome and mild neurofibromatosis). Congenital scolioses and those with dystrophic bone changes (dystrophic neurofibromatosis, osteogenesis imperfecta, skeletal dysplasias) are much more rigid and, thus, two stage surgery is usually necessary.

Severe idiopathic curves usually imply an early onset, but if encountered during the early years of life the application of a corrective cast may either prevent progression or, indeed, favour resolution, presumably because of the greater malleability of the young skeleton. If, however, progression continues, surgical treatment at a very young age is both necessary and very problematic (see below, page 522).

Idiopathic kyphosis (Scheuermann's disease), is uniplanar and rotationally stable and therefore eminently suitable for conservative treatment – any extension device can lead to a true physiological correction. Provided there is growth potential in the anterior part of the epiphyses, correction will occur, and if the spine does not stop growing till the middle of the third decade, conservative treatment (an extension brace) should be prescribed until at least the early twenties. This applies to all other kyphoses where there is anterior growth potential (some mucopolysaccharidoses, skeletal dysplasias, mild neurofibromatosis). Only

paralytic spines and had the added advantage that the system was sufficiently strong to obviate postoperative support. Soon after its introduction, Luque's wiring was used for idiopathic scoliosis, but curve correction was less good than that obtained by Harrington instrumentation, and there was a high rate of neurological complications associated with passing wires close to the dura.

For thoracic and thoracolumbar curves, derotation can only be effectively performed if the rod is bent to conform with a normal thoracic kyphosis, rod rotation being prevented by a square-ended lower hook-rod relationship. The segmental wires need only be placed under the laminae on the concave side, which reduces the neurological risk, and when these are lifted posteromedially all three components of the deformity are significantly improved (Figure 11 – before and after instrumentation).

For the flexible curve this can be performed in one posterior stage, but

with more severe curves, rigidity prevents adequate correction and if the lordosis is to be drawn back into a kyphosis, the spinal cord may come under undue tension. Some form of anterior shortening procedure is, therefore, necessary and for moderately rigid curves (Cobb angle 60° – 90°) this can be achieved by multiple anterior discectomies performed through a standard thoracotomy on the convex side. The posterior wiring is then carried out in a second stage some 2–3 weeks later.

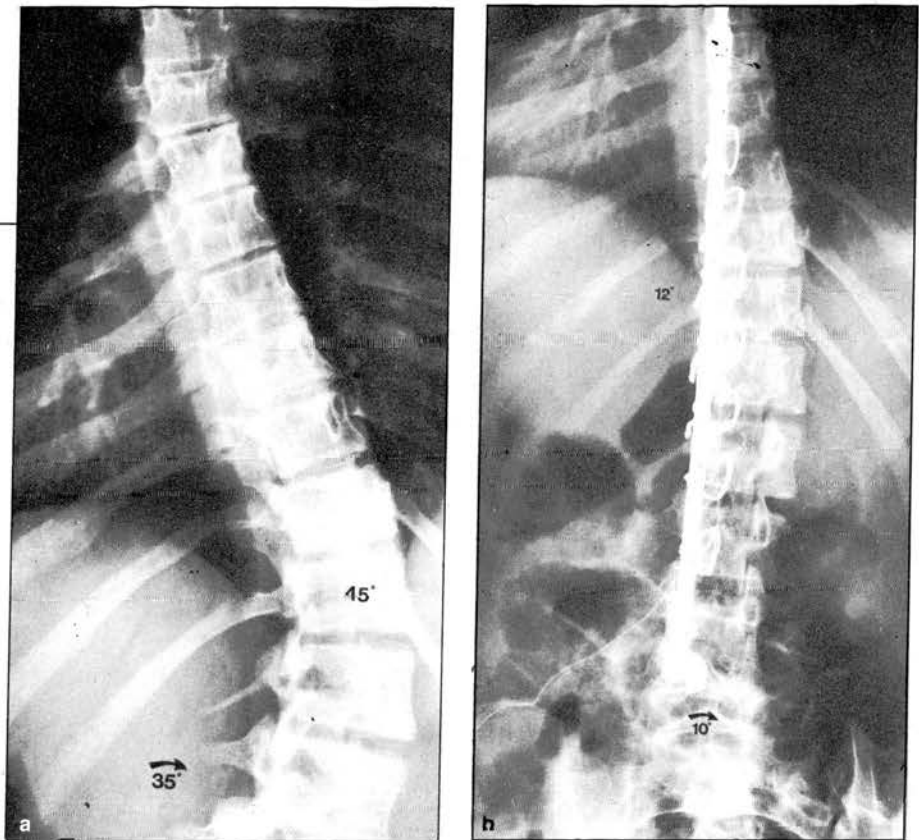
For the most rigid curves (Cobb angle of 90° or more) even anterior discectomy does not provide sufficient mobility and it is necessary to shorten the spine by removing a convex based wedge of bone from the apex of the curve. This is carried out in two stages, taking the anterior vertebral body first and then the posterior elements to complete the wedge in a second stage 2–3 weeks later. The wedge is closed using a Harrington compression system on

if anterior growth is not forthcoming or does not exist (congenital kyphoses and severe neurofibromatosis) is surgical treatment always necessary. Correction of an unacceptable kyphotic deformity also requires anterior surgery in the form of disc or bone removal and a firm strut graft (Figure 13). This can be followed by second stage posterior spinal instrumentation and fusion to give added support.

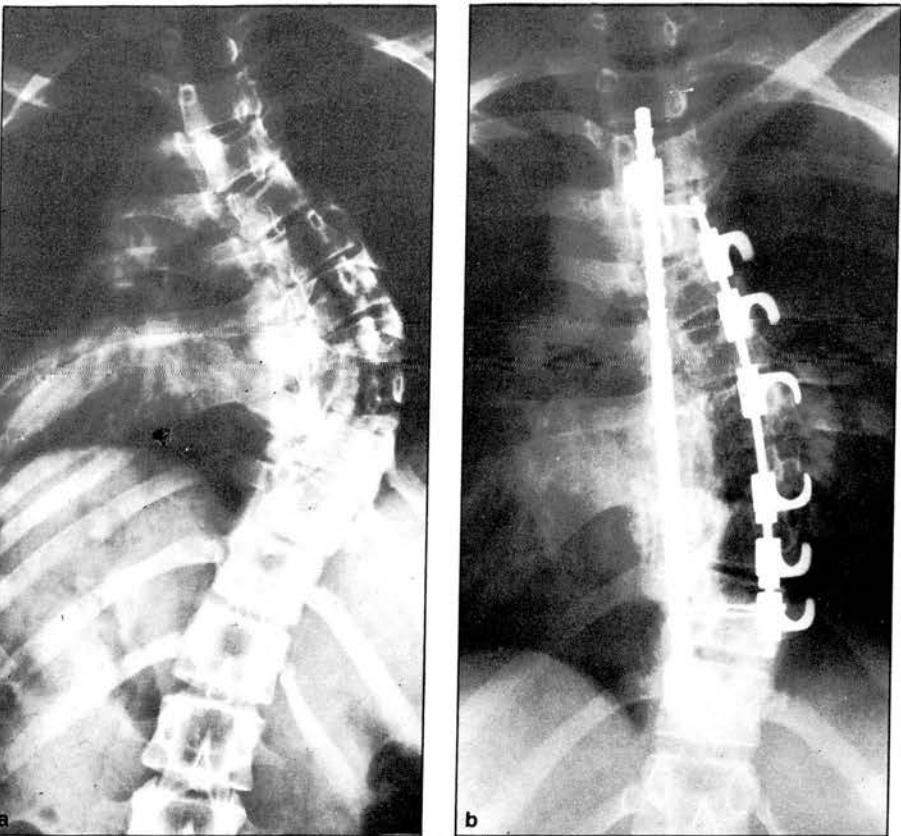
Potential progression

When the deformity has been 'corrected', the spine must be stabilized so that the deformity does not recur with subsequent growth. This is the reasoning behind spinal fusion. For late onset deformities, whose progression potential is necessarily limited, this can be performed posteriorly. The posterolateral elements are decorticated and allograft cancellous bone is added 'in profusion'. It is not necessary to use autogenous iliac crest bone and therefore a longer operation with more scarring, blood loss and postoperative pain is avoided. This is important in conditions such as osteogenesis imperfecta where there is insufficient autogenous bone anyway.

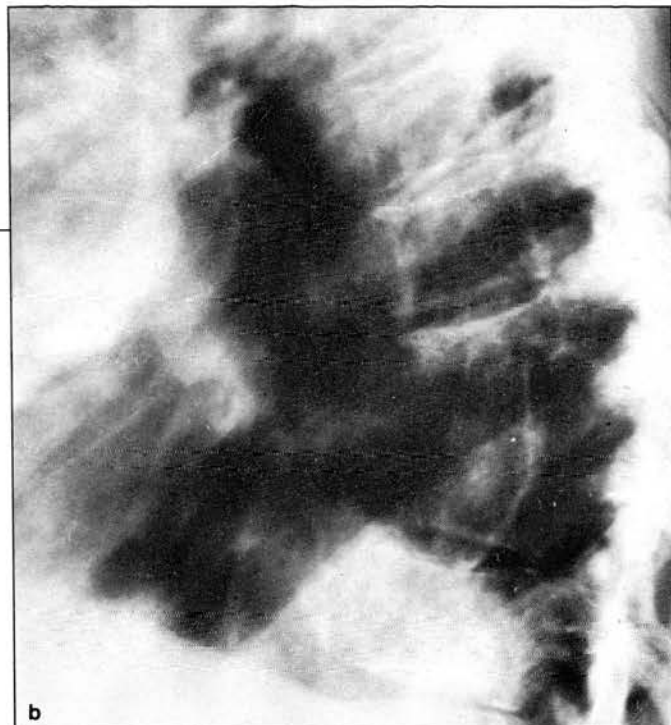
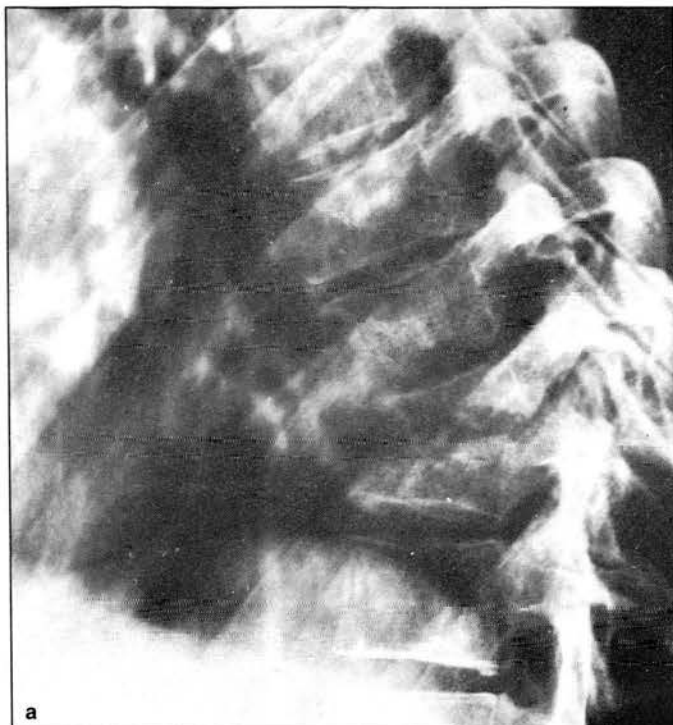
Because these deformities are lordotic, tethering the back of the already too short spine, may, in fact, add to the deformity; this happens if a posterior fusion is performed on an early onset case. To prevent progression with some reliability for the early case, the whole of the deformity must be set solid in bone by both anterior and posterior fusions. Spinal growth is significantly impeded by this total fusion, but it should be remembered that most of the growth of the spine has been completed by 10 years of age and that in the conditions for which this is necessary (early onset idiopathic scoliosis, congenital deformities, osteogenesis imperfecta, and skeletal dysplasias) short stature is the norm.



11 PA radiograph of an idiopathic spinal deformity before (a) and after (b) unilateral segmental spinal instrumentation.



12 PA radiographs of a patient with a severe rigid deformity before (a) and after (b) two-stage wedge resection.



13 Lateral radiographs of a patient with spinal tuberculosis before (a) and after (b) anterior vertebral body resection and anterior strut grafting.

Most cases are, however, rigid, and through the necessary two-stage wedge resection procedure the apical deforming mechanism is removed (e.g. unilateral failure of segmentation or dystrophic vertebra) and, thus, only a limited anterior fusion at the site of resection is necessary. For the less severe, early onset case, growth can be preserved by segmental spinal instrumentation without fusion, in the hope that the spine can be allowed to grow in a less deformed manner as the segmental wires migrate down the longitudinal rods. This is only a temporary measure, but the longer the definitive fusion is withheld, the more spinal growth will occur and the less likelihood there will be of fusion tethering.

Paralysis

Paralysis can cause spinal deformity and some spinal deformities may cause paralysis. The indications for surgery in the paralytic spine are twofold – loss of stability when sitting or loss of the potential for walking as a direct result of the spinal deformity. Paralytic deformities collapse under gravitational load and both anterior and posterior instrumentation and fusion may be required to ‘prop them up’. Anterior instrumentation (Dwyer or Zielke) is inserted after multiple anterior discectomy

throughout the curve. Cancellous screws are passed transversely across the vertebral bodies from the convex side and holes in the screw heads receive either a cable or threaded rod. When the screw heads are approximated by tightening the cable or rod, both correction and fusion are produced. In the second stage, standard posterior Harrington instrumentation and posterolateral fusion are performed.

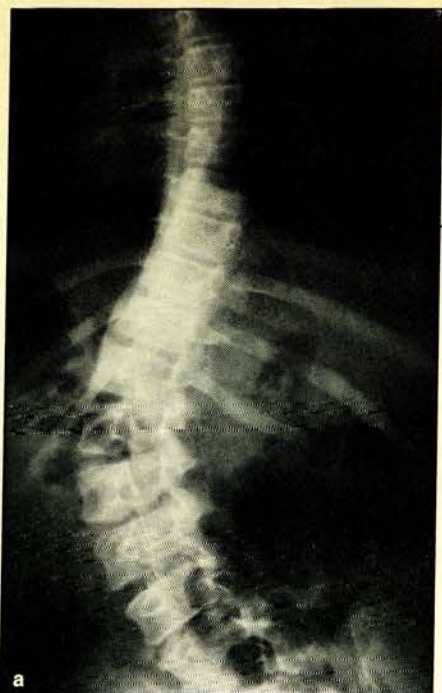
This procedure involves a considerable amount of surgery and some form of postoperative immobilization (bed rest or cast). For less severe paralytic curves one-stage posterior segmental spinal instrumentation will suffice and immobilization is not generally necessary (Figure 14).

Congenital kyphoses, severe Scheuermann’s disease, the dystrophic kyphosis of Von Recklinghausen’s disease, the kyphoses of tumours and infections can all give rise to spinal cord pressure, usually as a result of the spinal cord being drawn tight over the back of the kyphotic apex, though with tumours or infection, it can also be the result of direct pressure extensions of those pathological processes. While it might be expected that improvement of the degree of kyphosis by anterior discectomy above and below the

apex, with the insertion of distraction strut grafts, would improve the situation, anterior vertebral body resection at the apex of the curve is the only sure method of decompressing the cord. The defect is then supported by a strut graft of iliac crest bone. Laminectomy should *never* be performed because the anterior pressure on the cord cannot be relieved and the stability of the posterior elements is disrupted.

The underlying condition

The underlying condition is important in that the spinal deformity must not be dissociated from its other, often more important, features. Thus, while it is appropriate to treat non-progressive muscular dystrophies, debate rightly exists about whether the collapsing spine of the always fatal Duchenne dystrophy should be treated at all. Similarly, while the risks of operating upon the spinal deformity associated with Marfan’s syndrome can be accepted, the strong likelihood of arterial and venous thrombosis in homocystinuria, and the excessive skin fragility and tendency to uncontrollable haemorrhage in the Ehlers-Danlos syndrome, totally eliminate the possibility of surgery. The natural history of the underlying condition is a most important consideration. For



14 PA radiographs before (a) and after (b) bilateral segmental spinal instrumentation for a neuromuscular spinal deformity.

example, while the thoracolumbar kyphosis of Morquio's syndrome and spondylo-epiphyseal dysplasia should be extension-braced, 90% of the thoracolumbar kyphoses in achondroplasia resolve spontaneously and, therefore, the opportunity for resolution should be provided before treat-

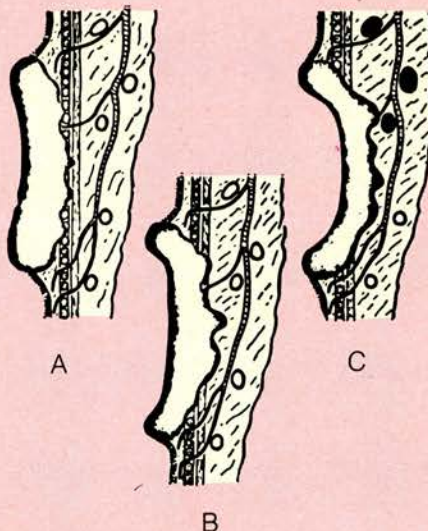
ment is considered. The spine in osteogenesis imperfecta is so porotic that instrumentation is particularly difficult, even with the use of methyl methacrylate cement; if there is a risk that 'more harm will be done than good', surgical intervention should be withheld. □

Cuthbert Esquire Dukes 1890-1977

H Brendan Devlin MD FRCS FRCS(I)

Cuthbert Dukes was appointed to St Mark's Hospital, London in 1922 and with a grant from the British Empire Cancer Council he headed the new pathology and research department there in 1924. He was responsible for many advances in the surgical pathology of the colon and rectum including divining the association between ulcerative colitis and cancer (1952) and describing the natural history of familial polyposis of the colon (1956).

Dukes' classification of rectal tumours (1936) - 'A' cases are those in which carcinoma is limited to the wall of the rectum, there being no extension into the extrarectal tissues, and no metastases in the lymph nodes. 'B' cases are those in which the carcinoma has spread by direct continuity to the extrarectal tissues, but has not yet invaded the regional lymph nodes. 'C' cases are those in which metastases are present in the regional lymph nodes. This method of classifying rectal can-



Surgery HISTORICAL VIGNETTE

Rectal tumours - Dukes' classification

cers for prognostic purposes was a pathological adaptation of the clinical grouping of operative specimens originally described by J P Lockhart-Mummery in 1928. Dukes reported that of the cases of rectal cancer coming to surgery, 15% were A cases, 35% B cases and 50% C cases. In 1935, in a further paper, Dukes revealed the results of dissecting the nodes in the resected specimen allowing the C cases to be subdivided into C1 cases, in which one or more non-malignant glands were present in the upper vascular pedicle, and C2 cases, in which malignant deposits were present in the glands up to the level of the ligation of the blood vessels. The 5-year survival after treatment for rectal cancer on the Dukes' classification is A cases - 90%, B cases - 70%, C cases - 30%; the overall 5-year survival of all cases aggregated is 50%.

A common modification of Dukes' classification is the addition of a D category to signify those patients who at operation were noticed to have distant (usually liver) metastases.

FURTHER READING

- Deacon P, Flood BM, Dickson RA. Idiopathic scoliosis in three dimensions. A radiographic and morphometric analysis. *J Bone Joint Surg (Br)* 1984; **66B**: 509-12.
- Dickson RA. Editorial - screening for scoliosis. *Br Med J* 1984; **289**: 269-70.
- Dickson RA, Bradford DS. *Modern management of spinal deformities*. London: Butterworths, 1984.
- Leatherman KD, Dickson RA. Two-stage corrective surgery for congenital deformities of the spine. *J Bone Joint Surg (Br)* 1979; **61B**: 324-8.
- Luque ER. The anatomic basis and development of segmental spinal instrumentation. *Spine* 1982; **7**: 256-9.
- Roaf R. The basic anatomy of scoliosis. *J Bone Joint Surg (Br)* 1966; **48B**: 786-92.
- Roaf R. *Scoliosis*. London: ES Livingstone, 1966.

CROSS REFERENCES

- Cerebral palsy: *SURGERY* **12**, 287.
- Poliomyelitis: *MEDICINE International 2nd series* **3**, 91.
- The early management of spina bifida: *SURGERY* **6**, 137.
- The malformed child - chromosomal abnormalities and dysmorphic syndromes: *MEDICINE International 1st series* **36**, 1976.
- Thoracotomy: *SURGERY* **2**, 50.

Vesico-ureteric reflux

Radiographic investigation of children with urinary tract infection reveals vesico-ureteric reflux (VUR) in 30–50% of cases. At any age, reflux is abnormal; the incidence is higher in girls and on the left side, and there is often a familial tendency.

Pathological effects

The intramural course of the normal ureter (Figure 1) prevents VUR by acting as a passive and dynamic sphincter. Incompetence of the ureterovesical junction allows organisms entering the bladder to gain access to the upper urinary tract and cause acute pyelonephritis. Healing of this bacterial infection leads to the typical radiological appearance of chronic pyelonephritis (reflux nephropathy)

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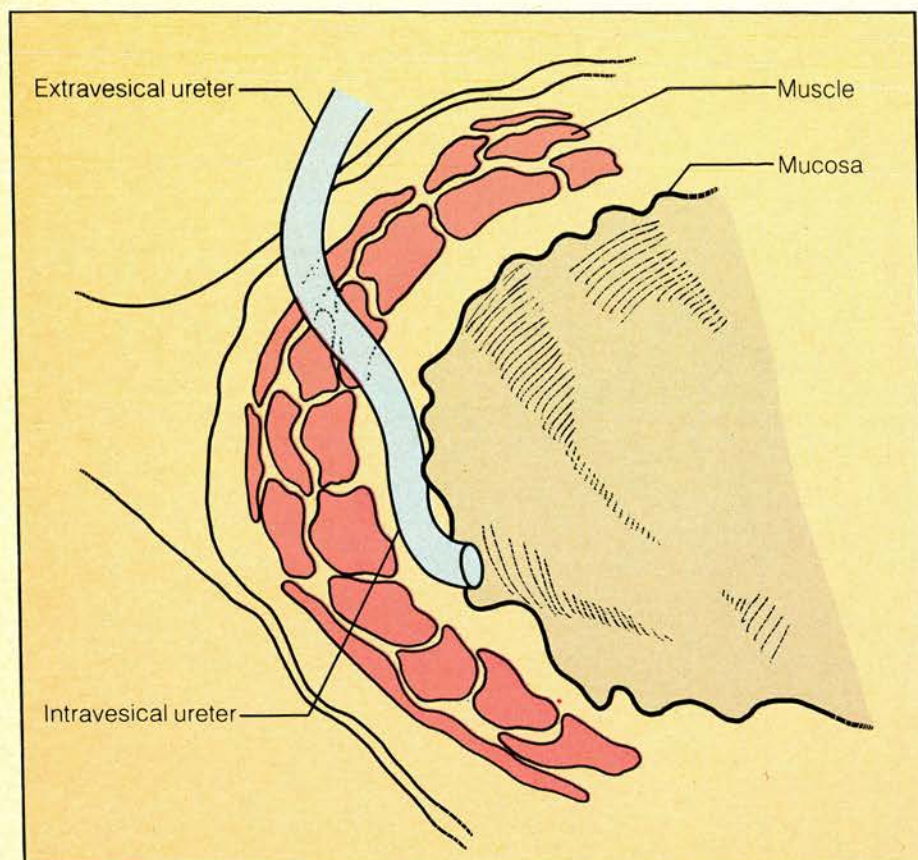
with clubbed and distorted calyces and loss of renal substance (Figure 2). When bilateral and severe, chronic pyelonephritis may lead to renal failure. The incidence of hypertension in patients with renal scars secondary to reflux is 10% over a 10-year

period; it may be higher over a longer follow-up time.

Physiological effects

In health, the renal pelvis intraluminal pressure seldom rises above 4 cm H₂O. Ureteric peristalsis may produce pressures of 25 cm H₂O but the renal pelvis is protected from these alterations of pressure by the competence of the pelvi-ureteric junction. High bladder voiding pressures (40–60 cm H₂O) are not transmitted to the upper urinary tract because of the normally competent ureterovesical junction.

Minor grades of reflux cause little measurable change in renal function, but after successful reflux surgery, an improvement in renal function



1 The intramural course of the ureter acts as a static and dynamic sphincter to prevent vesico-ureteric reflux.

Causes of vesico-ureteric reflux

Genetic

- Familial
- Lateral ectopia of ureteric orifice
- Duplex pelvicalyceal collecting system
- Para-ureteric diverticula

Obstruction

- Neuropathic bladder
- Posterior urethral valves

Associated with congenital anomalies

- Renal hypoplasia/dysplasia
- Renal agenesis
- Renal ectopia – fusion, horseshoe kidney
- Pelvi-ureteric junction hydro-nephrosis
- Anorectal malformations

Postoperative

- Decompression of a ureterocele
- Release of a stone

Mega-ureters

- Refluxing
- Obstructive

Idiopathic

- Sphincter dysinergia
- Infection – primary or secondary
- Constipation

OPERATIVE SURGERY FOR SPINAL DEFORMITY.

Dickson RA, Deacon P, Archer IA.

In 'Current Operative Surgery: Orthopaedics and Trauma'
(Hughes SPF Ed) Balliere Tindell: London etc, 114-146, 1985.

This chapter described in detail the pathogenesis of spinal deformities, the indications for surgery and patient selection, and the various techniques that are necessary to correct deformities of different types and magnitudes.

Operative Surgery for Spinal Deformity

Robert A. Dickson, Phillip Deacon and Ian A. Archer

INTRODUCTION

In order to appreciate what must be done operatively to correct a spinal deformity it is necessary to fully understand the nature of these three-dimensional deformities and their pathogenesis. Spinal deformities fall into two broad categories—scoliosis and kyphosis. It will be seen that both these deformities stem from a disorder of spinal shape in the sagittal plane and that one, scoliosis, is associated with rotation and is therefore a complex three-dimensional deformity, while the other, kyphosis, is protected from rotation and exists in only one, the sagittal, plane.

Normal spinal shape

The spine is supposed to be straight in the coronal plane, although school screening studies show an enormous number of children with minor degrees of coronal plane asymmetry.¹ Scoliosis refers to an abnormal amount of curvature in the coronal plane. In the sagittal plane four primary curvatures are recognized from the time the child assumes the upright posture (Figure 7.1). Lordoses exist in the cervical and lumbar regions while kyphoses exist in the thoracic and sacral regions. The axis of spinal rotation runs through these normal sagittal curves so that the cervical and lumbar lordoses are in front of the axis while the thoracic kyphosis is behind it. Areas of lordosis, particularly on forward flexion, are rotationally unstable, there being too much vertebral column material anteriorly to be accommodated without rotating to the side.² By contrast the thoracic kyphosis will be



Figure 7.1 The four primary sagittal curves and their relationship to the axis of spinal rotation—the lordotic cervical and lumbar regions lie in front of the axis in contrast to the kyphotic, thoracic and sacral regions, which lie behind it.

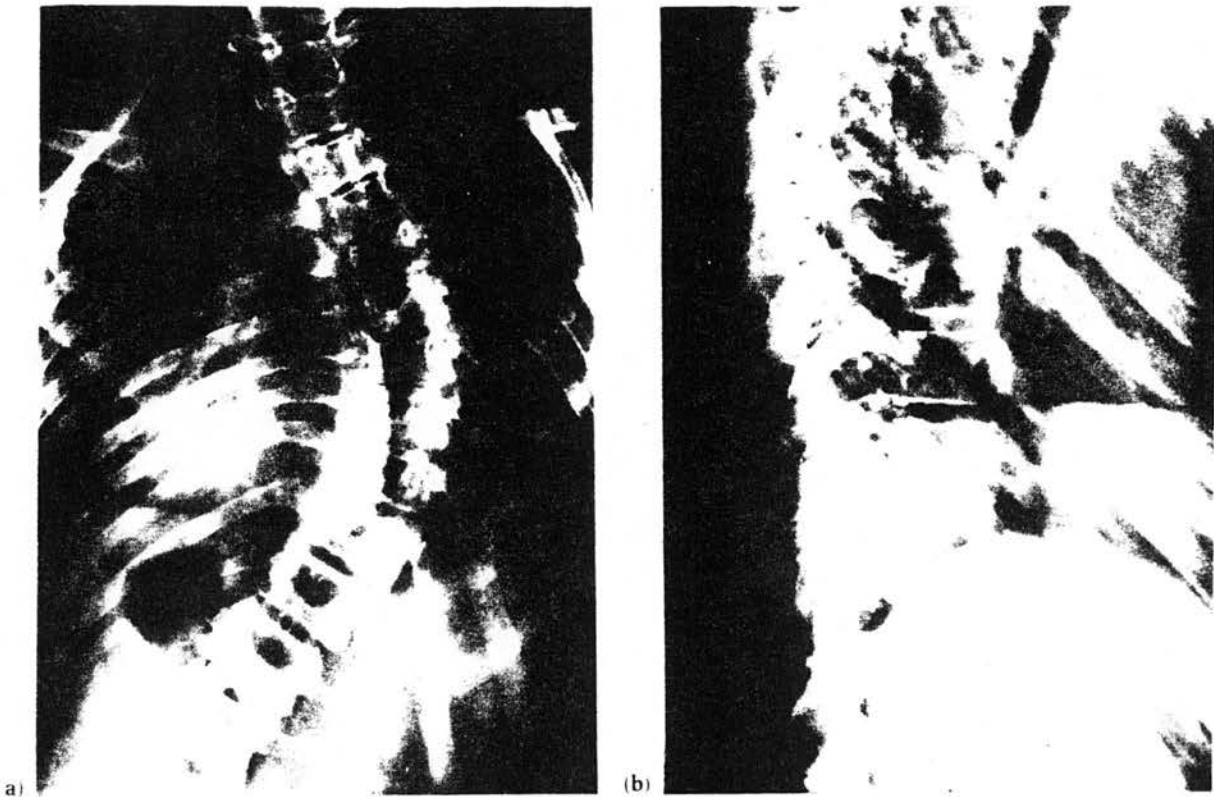


Figure 7.2 (a) A posteroanterior (PA) radiograph of an idiopathic scoliosis—there is a lateral spinal curvature with vertebral rotation. (b) True lateral radiograph of the apex of the same curve showing the lordosis. (c) (see over) Posteroanterior radiograph of a congenital scoliosis showing multiple rib and vertebral anomalies. (d) (see over) True lateral radiograph of the same curve. Again there is an underlying lordosis.

(For a better quality picture see Publication 19, Figures 1 and 2.)

protected from rotation by its anteriorly situated axis. Accordingly, in areas where a lordosis normally exists there are powerful inbuilt protection mechanisms which resist rotation under normal circumstances.³ First, there is much available intersegmental flexion movement so that the limits are seldom, if ever, reached. Second, the broad side-to-side width of the cervical and lumbar vertebral bodies protects against rotation on forward flexion. Third, behind these lordoses exist the most powerful soft tissues anywhere in the spine (such as ligamentum nuchae, interspinous and supraspinous ligaments and erector spinae muscle). In the thoracic region the vertebrae are heart-shaped with a more pointed anterior aspect and this transverse plane configuration is rotationally unstable. It is therefore protected by the presence of a kyphosis so that the anterior aspects of the thoracic vertebral bodies never find themselves under compression and thus do not rotate. There is very

little intersegmental flexion available, and little protection from posterior soft tissues, and thus if the thoracic kyphosis should be lost at any level, then rotation is inevitable.

Production of scolioses

Non-structural scolioses (for instance, those that exist in association with a leg-length inequality or above or below a structural curve) exist in the coronal plane with little or no rotation, unless they are in the lumbar region where there already is a lordosis, and certainly no progression potential.⁴ By contrast, structural scolioses have both rotation and progression potential and it is the nature of this rotational process which holds the key to the understanding of the pathogenesis of these scolioses and thus their optimal correction. If the mechanisms mentioned above which support normal spinal shape in the sagittal plane fail, then a



(c)



(d)

(See p. 115 for caption.) (For a better quality picture see Publication 35, Figure 1.16.)

progressive spinal deformity can be produced.³

The great majority of spinal deformities are idiopathic (self-generating), there being no obvious precipitating cause. These exist in the thoracic, thoracolumbar or lumbar regions and more than one curve may exist in the same spine. Regardless of where the idiopathic curve is, rotation is always in a constant direction, with the spinous processes directed towards the curve concavity and the vertebral bodies directed towards the curve convexity.

It is elementary geometry therefore that the deformity of idiopathic scoliosis must be lordotic throughout, the front of the spine being longer than the back (Figure 7.2). In the lumbar region there is already a lordosis as a normal feature and therefore an increased lordosis, a reduction of intersegmental flexion or posterior stiffness, must be superadded to produce an environment-favouring rotation. In the thoracic region quite a radical alteration in sagittal spine shape is necessary because the thoracic kyphosis must be reversed

into a lordosis. Then the anterior borders of these heart-shaped vertebrae lie in front of the axis of rotation, undergo compression on forward flexion and rotate to the side to be accommodated (the idiopathic rib hump is increasingly more obvious on forward bending).

These same features also produce rotational scolioses from a primary lordosis in 'idiopathic-type' deformities encountered in association with other conditions. It is the nature of the condition (bone weakness in neurofibromatosis and osteogenesis imperfecta, and soft tissue weakness in Marfan's syndrome, Ehlers-Danlos syndrome and homocystinuria) which makes the idiopathic type rotated lordoscoliosis more prevalent than in the 'normal child'. The important point is that it is the same

deformity type and thus requires the same correction strategy. In severe osteogenesis imperfecta and neurofibromatosis, where there is clear evidence of dystrophic bone change, then the rotated lordoscoliosis can be quite sharp and angular. Congenital bony deformities (hemivertebrae and unilateral bars) produce their deformities in precisely the same way, although with hemivertebrae the deformity tends to be mostly in the coronal plane with little evidence of rotation while with unilateral bars the posterior elements of the spine are commonly tethered to produce a significant lordosis and thus a rotational potential (see Figure 7.2).

With myelodysplasia and neuromuscular deformities of the spine it is the spinal musculature

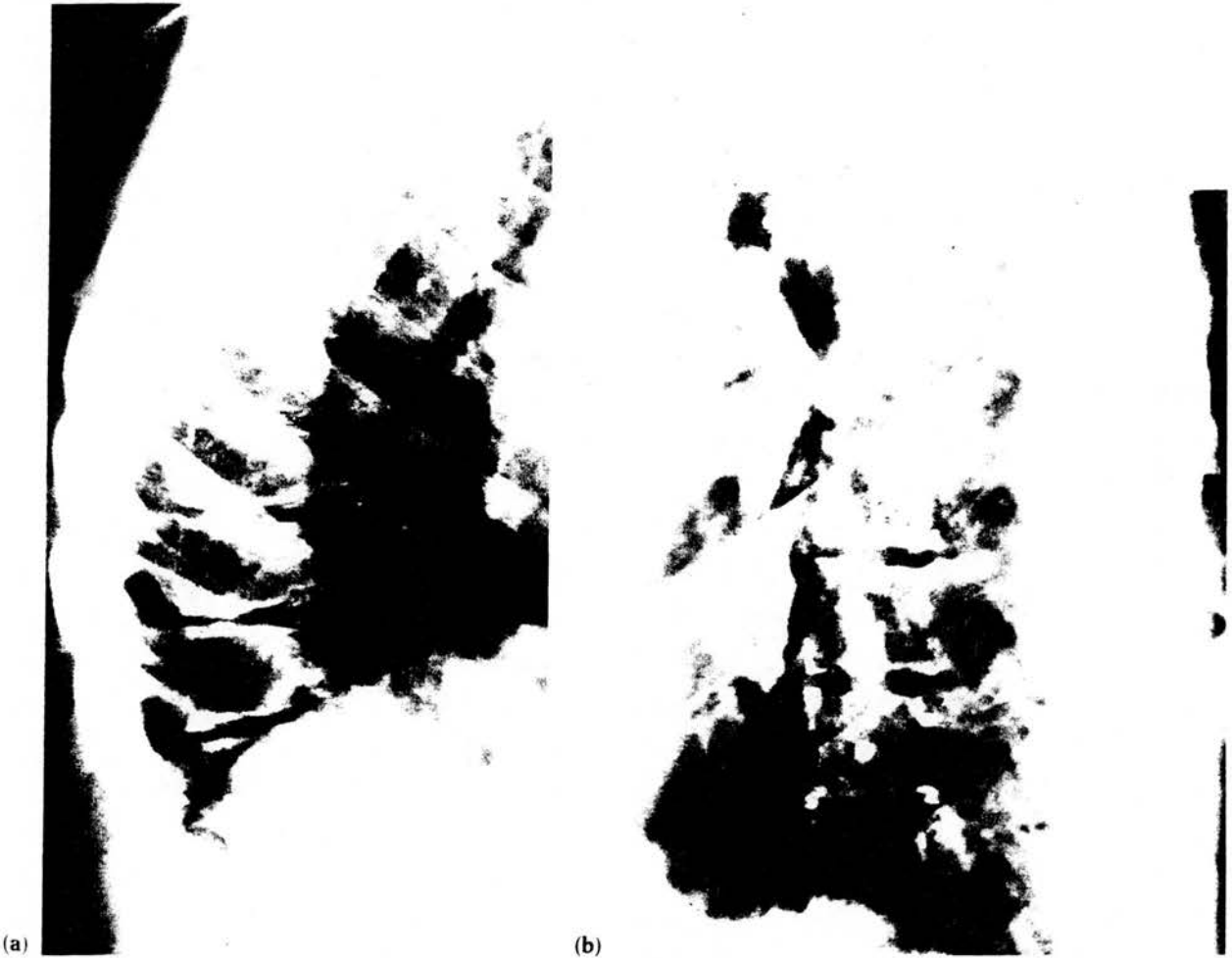


Figure 7.3 Composite lateral radiographs of (a) idiopathic scoliosis (left) and (b) Scheuermann's disease (right). In idiopathic scoliosis the vertebral bodies are wedged with anterior height greater than posterior height, exactly the opposite situation to that in Scheuermann's disease where anterior height is less than posterior height.

(For a better quality picture see Publication 26, Figures 3d and 4.)

behind the existing lumbar lordosis which fails, to produce the typical collapsing paralytic lordoscoliosis. Increasing body weight and loss of trunk balance contribute to the associated pelvic obliquity. Therefore, although the mechanism of production is different from idiopathic-type deformities and congenital deformities, and a different treatment strategy will be required, it is still the same deformity that needs to be treated—in other words, a lordoscoliosis.

Production of kyphoses

The great majority of pathological kyphoses encountered exist in the thoracic region and are therefore an exaggeration of normal spinal shape in the sagittal plane. As typical thoracic vertebrae

are kyphotically wedged by 3° and Scheuermann's disease is considered to exist if kyphotic wedging is 5° or more,⁸ then there does not have to be a great change in sagittal spine shape in the thoracic region to produce a pathological kyphosis. Scheuermann's disease is of course the commonest type of pathological kyphosis encountered and is the opposite deformity to idiopathic scoliosis (Figure 7.3). Being rotationally stable, with the axis of spinal rotation anterior to the kyphotic vertebrae, there is no associated scoliosis unless the kyphotic process is slightly asymmetric (the kyphotic wedging has affected more the right than the left side of the spine). In this situation a mild local scoliosis can occur but with the spinous processes rotated towards the curve convexity as would be expected with an asymmetric kyphosis.

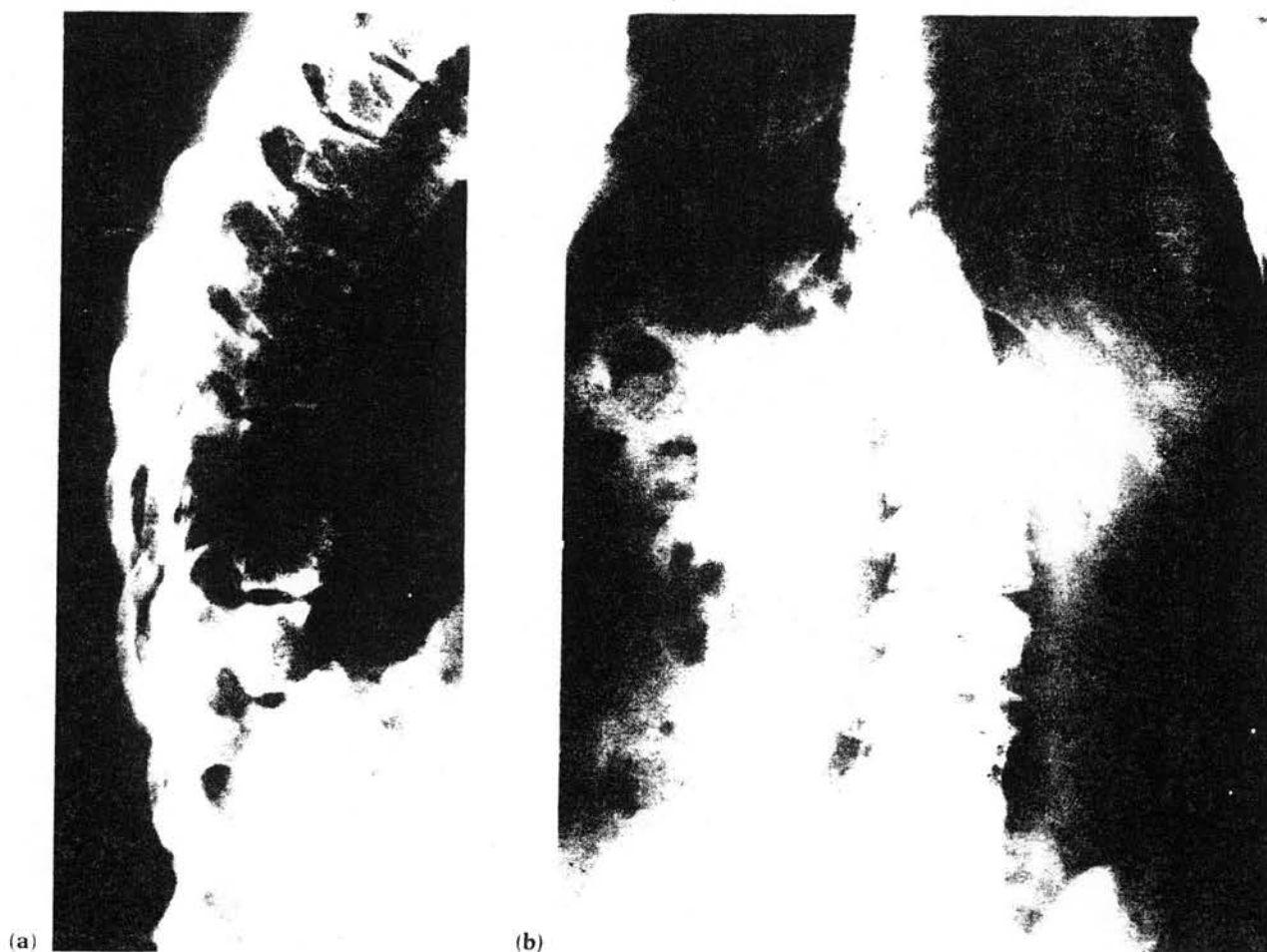


Figure 7.4 (a) Lateral radiograph of a patient with Scheuermann's disease—there is a region of compensatory lordosis below the Scheuermann's kyphosis. (b) Posteroanterior radiograph of the same patient showing a mild idiopathic scoliosis corresponding to the lordotic region below the level of the Scheuermann's kyphosis.

(For a better quality picture see Publication 14, Figures 2 and 3.)

While Scheuermann's disease is produced by epiphyseal growth being relatively reduced anteriorly, other pathological kyphoses are produced by conditions which destroy bone in the post-mature phase. Thus tumours and infections are common causes of kyphosis, invasion of bone by neoplasm or sepsis causing structural failure. When tumour or infection exists in the cervical or lumbar regions, which are normally lordotic, quite a radical change in shape has to occur before there is a frank kyphosis. Thus it can be seen that because scolioses and kyphoses are opposite deformities in the sagittal plane, the deformity of kyphoscoliosis does not really exist. It is however possible for a kyphosis to exist in one part of the spine and a scoliosis to exist above or below it, and this is encountered in more than 50% of patients with Scheuermann's disease (Figure 7.4), but this should not be called kyphoscoliosis as this term implies that the deformities coexist at the same site.

As most spinal injuries are by way of vertical loading in flexion the traumatic kyphosis is the commonest deformity produced. Moreover, because there may be associated neurological damage it is not uncommon to encounter an area of kyphosis above an area of paralytic rotated lordoscoliosis. If the local area of traumatic bony injury has been further insulted iatrogenically by laminectomy, the bony kyphosis can progress rapidly.

INDICATIONS AND PATIENT SELECTION

The management philosophy of spinal deformities hinges on four considerations: size of the deformity at presentation, progression potential, the presence of paralysis, and the underlying condition.

Curve size at presentation

If the deformity is acceptable at presentation, preservation of acceptability is the aim, although there is no convincing evidence that any form of non-operative treatment alters the natural history of a scoliosis.⁶ If the deformity is unacceptable, it must be made acceptable once more and this is the aim of surgical treatment. Great reliance has been placed upon the use of Harrington instrumentation over the past 25 years, principally for idiopathic and idiopathic-type deformities, despite the fact that the instrumentation was designed to prop up the poliomyelitis scoliosis. A cursory glance at the lordoscoliotic nature of the idiopathic deformity indicates that the Harrington distraction rod, which

is hooked into the spine above and below the curve, cannot achieve any correction of rotation, and this is borne out by recent studies.⁷ This is a serious matter, as the great majority of spinal deformities which require treatment are the late-onset idiopathic type which are solely a question of deformity, of which the rotational prominence is the presenting complaint.

The Harrington compression system is an optional extra which certainly aids stability but probably does not materially alter the degree of correction. While Harrington instrumentation may favourably influence the small flexible curve that has just reached unacceptability it has to be said that such circumstances at presentation are few and far between and the 'typical adolescent case' has much more rotation at presentation than can be satisfactorily corrected in the traditional way (Figure 7.5).

A recent important advance in the treatment of spinal deformities has been the introduction of segmental spinal instrumentation.^{8,9} Two metal rods are laid along each side of the spine, pre-bent to the desired correction, and then the spine is wired to these rods at each vertebral level. The wires are passed under the laminae on each side, which is the firmest part of the vertebral arch. This instrumentation knows its best indications in scolioses of neuromuscular origin and it has quite rightly been considered too dangerous to use for the otherwise uncomplicated idiopathic case. Certainly the use of two sets of rods and wires is overinstrumentation. However, remembering the three-dimensional nature of the rotated lordoscoliosis, derotation can be achieved by using sublaminar wires only on the concave side of the spine and by tightening these wires round a Harrington distraction rod; this will elevate the depressed concavity and thus derotate. The presence of a straight Harrington rod may allow derotation if the lordosis is big enough but more usually the rod has to be bent into some degree of kyphosis, rotation of which is prevented by the use of a square-ended lower hook, so that satisfactory derotation takes place.^{3,10} In the lumbar region it is important that the lumbar lordosis is not obliterated by the use of a straight rod as this leads to an unsightly appearance and gait pattern. The rod should therefore be bent into a mild degree of lordosis before the concave sublaminar wires are tightened round it.

In the thoracolumbar and lumbar regions the idiopathic lordoscoliosis can be very nicely corrected by the use of anterior instrumentation. This was developed by Dwyer¹¹ in Australia specifically for the insertion into a rotated lordoscoliosis, where

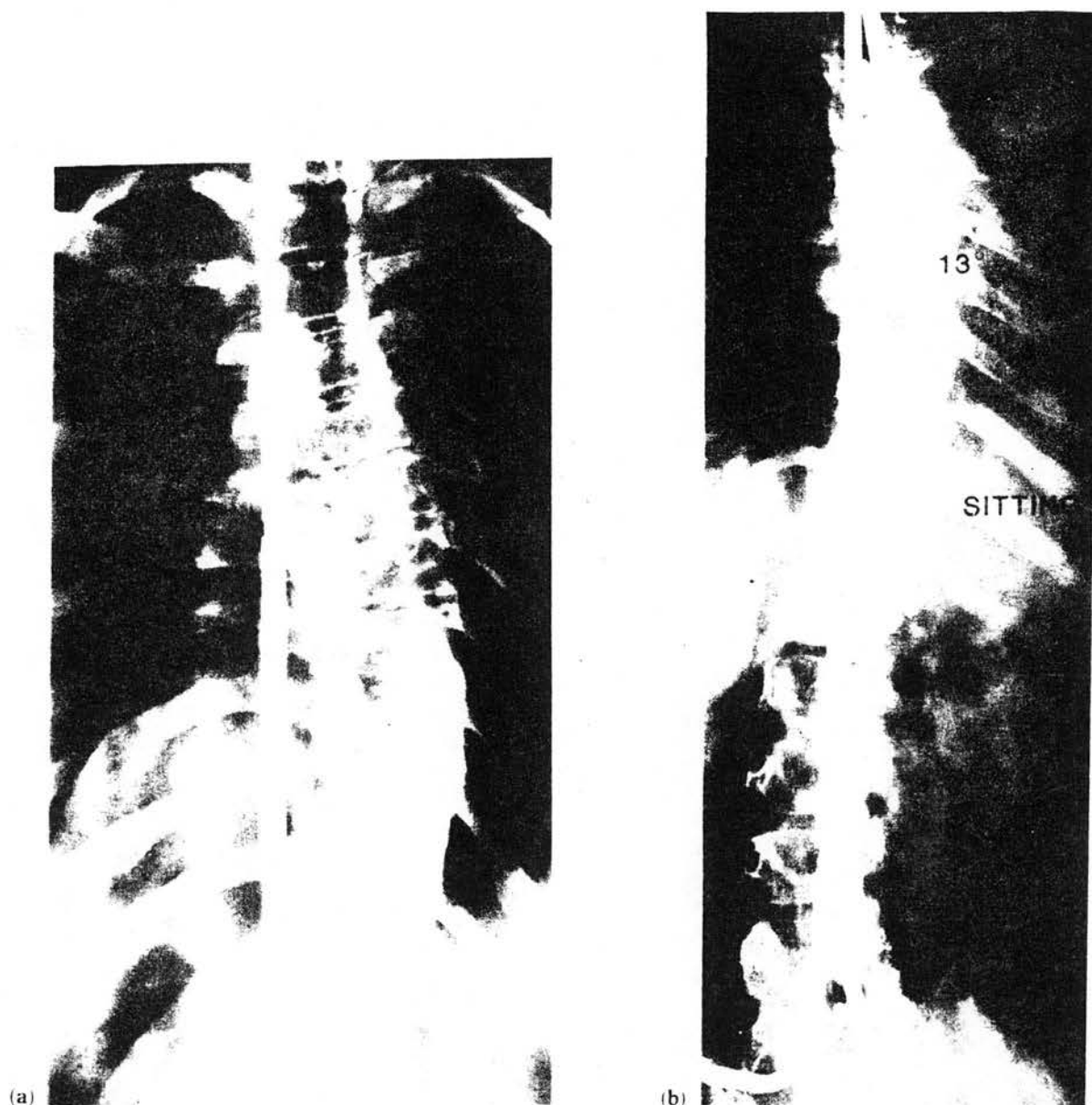


Figure 7.5 Posteroanterior radiographs of the spine after different types of instrumentation. (a) Harrington distraction instrumentation. (b) Luque instrumentation. (Reproduced by kind permission of Mr J. Dove, FRCS, and the Medical Illustration Department, Hartshill Hospital, Stoke-on-Trent). (c) Harrington-Luque instrumentation—a Harrington distraction rod is pre-bent to a kyphotic configuration and combined with segmental sublaminar wires. (d) Dwyer instrumentation. (e) (see p. 122) Zielke instrumentation. (Reproduced by kind permission of Mr J.K. Webb, FRCS, and the Medical Illustration Department, Harlow Wood Orthopaedic Hospital, Mansfield.)

(For a better quality picture of Fig 7(a) see Publication 35, Figure 4.17 and for Fig 7(b) see Publication 35, Figure 4.52.)



(c)

(For a better quality picture see Publication 35, Figure 4.54.)



(d)

(For a better quality picture see Publication 35, Figure 4.23.)



Figure 7.5(e) Zielke instrumentation (see pp. 120 and 121 for parts (a)–(d).) (For a better quality picture see Publication 35, Figure 4.24.)

the front of the spine is longer than the back. The anterior aspect of the spine is approached on the convex side by a thoracoabdominal approach and, after disc removal throughout the curve, screws are inserted transversely from the convex side across the vertebral bodies. The screw heads are cannulated to receive a cable which, when tightened, shortens the convex side and thus effects a straightening. This apparatus has been markedly improved by the design modifications of Zielke and the excellent German instrument-makers.¹² However, to what extent the anterior approach should be used for the uncomplicated idiopathic case is also of concern, particularly as thoracolumbar and lumbar scolioses seldom give rise to the same degree of rotational prominence as the comparably sized thoracic curve. Although Dwyer devised his instrumentation for the idiopathic case, it has found its real value in the management of the collapsing paralytic curve of neuromuscular origin which requires all the support it can get.¹³

An important concept in the surgical manage-

ment of spinal deformities is curve rigidity. With idiopathic deformities, this tends to go along with curve size: the bigger the curve the more rigid it is. With congenital bony deformities there is very little, if any, natural curve flexibility at any time. Thus, once the idiopathic deformity approaches a Cobb angle of about 70° , it is becoming both too big and too rigid to be handled by a posterior approach alone. This would appear to be a prime indication for spinal traction but no matter how that traction is applied (non-skeletal traction, halo-femoral traction, halopelvic traction or halosuspension) it does not improve curve flexibility^{14, 15} and, moreover, some of the more aggressive forms of traction have an unacceptable neurological or thrombotic complication rate.^{16, 17} If a curve of 70° will readily bend to 50° and no further, then this situation will not be improved by traction nor one-off operation. The patient would therefore be undergoing a significant operative procedure with a minimal improvement in the coronal plane component of the deformity and no improvement in rotation. If the combination of a Harrington dis-

traction rod and concave sublaminar wiring were imposed on this situation and, provided metal work or bone did not fracture in the tightening process, it might be possible to obtain a little better correction, but with the very serious risk of causing an unacceptable degree of spinal cord tension.

If an area of lordosis is being pulled back to the straight or beyond then the spine must lengthen in the process. Therefore for the 'adolescent with a bigger curve' a preliminary anterior shortening procedure is necessary (a) so that the spine will not be unduly lengthened, and (b) so that space is made available for the lordosis to move through.¹⁰ For the Cobb angle curve between 70° and 90° this can be performed by way of anterior discectomy (Figure 7.6), removing the apical five or six discs by a thoracotomy or thoracoabdominal exposure according to curve site. This is performed as a preliminary first stage and then the combination of Harrington rod and concave sublaminar wiring is performed in a second stage some 3 weeks later. Staged procedures are an important part of the operative management of spinal deformities and these have come to be recognized as being much safer than trying to perform all the necessary surgery at one sitting.¹⁸

For the severe rigid curve of whatever aetiology (idiopathic beyond 90°, congenital, von Recklinghausen's, etc.) the only safe method of obtaining curve correction without incurring the possibility of tension paraplegia is to shorten the bony spine at the same time as straightening it (see Figure 7.6). Here the technique of wedge resection of the spine developed by Leatherman in Louisville is invaluable.¹⁸ The necessary-sized wedge of bone based on the convex side is removed and then the wedge-shaped defect is closed using posterior instrumentation. Thus curve correction is obtained without lengthening the spine. If the spine is completely rigid throughout the curve, as is encountered in severe congenital lesions, and if there has been a previous posterior fusion, it would be tempting to perform multiple osteotomies of the too short posterior side of the spine and effect a correction by distraction instrumentation. While this technique does have its proponents,¹⁹ the principle of spinal lengthening is too risky^{20, 21} and should not therefore be accepted when excellent correction in both coronal and transverse planes accompanies the Leatherman two-stage procedure.

If after a surgical procedure there is still an unacceptable rotational prominence, caused by the convex ribs and not the spine itself, then a derotation costoplasty is the most useful procedure.²² The convex rib hump is resected subperiosteally

and each rib end is attached to the one lower down, thus reducing the rib hump. If necessary, the resected rib portions can be used to augment the depressed concavity.

Progression potential

Because spinal deformities can progress until the end of spinal growth (not usually achieved until the middle of the third decade) an important part of the management strategy is to prevent the corrected position from deteriorating during the remainder of growth. Different curve types have different severities of progression potential, which is most important to recognize so that the correct procedure can be selected. Although instrumentation is able to resist progression to some degree, the definitive stabilization of the corrected position is achieved by spinal fusion.²³ It has been assumed that this can be achieved by a posterior approach and thus the operation of posterior spinal fusion is that traditionally recommended.^{24, 25} The situation is not so straightforward when the three-dimensional nature of the deformity is considered. These are lordoscolioses (see Figure 7.2), and the back of the spine is already too short. A posterior spinal fusion therefore will prevent it from becoming any longer with growth. Thus for the adolescent-onset idiopathic scoliosis with a milder progression potential a posterior fusion may in fact achieve the desired aim of preventing subsequent progression only because progression potential is minimal. However, for the early-onset progressive case quite the opposite is the situation and posterior fusion may cause accelerated progression by posterior tethering.^{2, 26-28} If the spine is to be fused anywhere, it ought to be the front, which is already too long in a lordoscoliosis. In practice both anterior and posterior fusions would be required for these very progressive cases.

Therefore when curve size at presentation and progression potential are put together, a management strategy for the great majority of spinal deformities is apparent. If the deformity is acceptable, but the natural history would indicate that it would become unacceptable, then no correction is necessary, merely stabilization by fusion or, experimentally, by instrumentation without fusion.¹⁰ If the progression potential is severe, the fusion must be anterior and posterior; if the progression potential is mild the fusion can be performed posteriorly to the great advantage of the surgeon. If the deformity is unacceptable at presentation then it should be corrected as outlined above before stabilization by fusion.

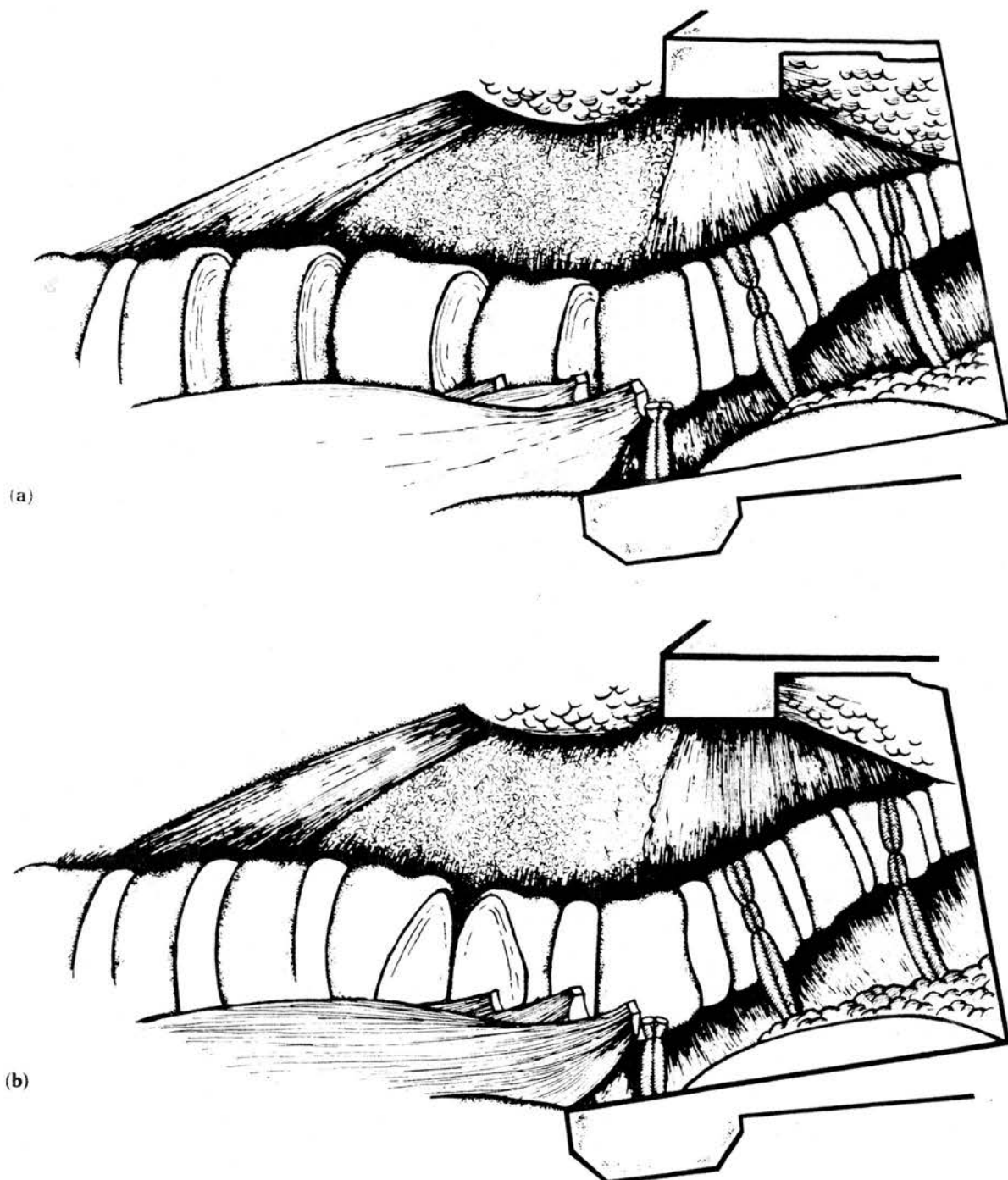


Figure 7.6 Two ways in which the front of the spine can be shortened: (a) anterior discectomy for moderately severe curves, (b) anterior wedge resection for severe rigid curves.

The presence of paralysis

When the spinal deformity occurs in association with impairment of the normal soft tissue support to the spine, a collapsing 'paralytic' C-shaped deformity occurs in the thoracolumbar region extending down to the sacrum, which is tilted so that the pelvis on the convex side of the scoliosis is low

(Figure 7.7). Conditions such as neuromuscular disorders, myelodysplasia and traumatic paralysis belong to this category, and here treatment is concerned with function. Threatened or lost ambulatory potential in the walker, or sitting stability in the sitter, are the indications for treatment, while cosmetic considerations are relegated. Although Harrington instrumentation was designed to prop

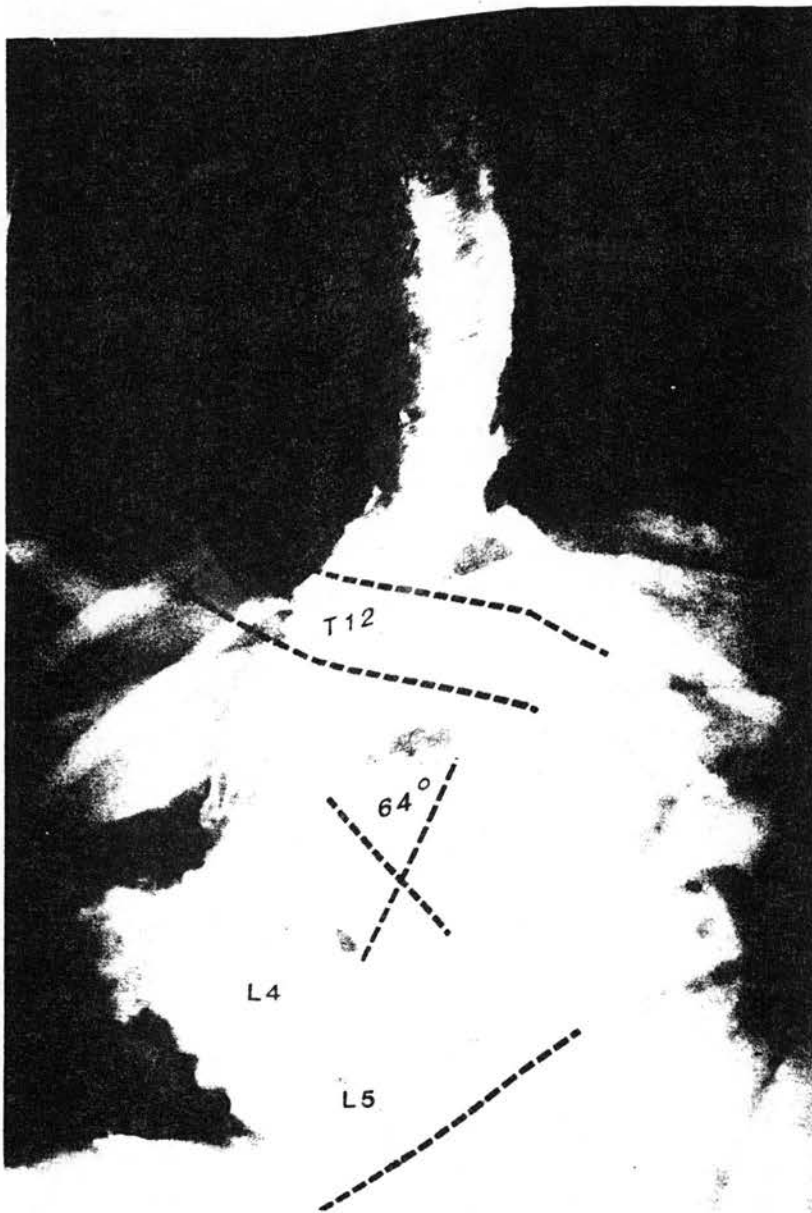


Figure 7.7 PA radiograph showing the typical 'paralytic' C-shaped collapsing lordoscoliosis with pelvic obliquity. (Reproduced by kind permission of Mr J. Dove, FRCS, and the Medical Illustration Department, Hartshill Hospital, Stoke-on-Trent.) (For a better quality picture see Publication 35, Figure 4.23.)

up the poliomyelitis collapsing curve.²⁹ it became clear that with such adverse mechanics the spine required to be instrumented and fused both front and back in order to avoid postoperative collapse.¹³ Thus the anterior instrumentation and fusion techniques of Dwyer¹¹ and Zielke¹² in the first stage, followed by posterior fusion with Harrington instrumentation in the second stage performed 3 weeks later, became the accepted practice for these curves.¹³

Recently Luque in Mexico demonstrated that by using metal rods and segmental sublaminar wires on both sides of the collapsing spine it was possible to both correct and maintain the corrected position in patients with poliomyelitis without the need for either a preliminary anterior stage or postoperative support (bedrest, plaster of Paris or brace).^{8,9} In practice this is only so for curves that are not significantly rigid. In this latter situation anterior shortening by way of discectomy or vertebral wedge resection and anterior instrumentation is necessary as a preliminary first stage. The use of the Luque instrumentation in the second stage, however, does facilitate much speedier rehabilitation.

The indications for the operative treatment of paralytic spine deformities must be remembered as being the consequences of the spinal deformity and not any central control impairment such as may exist with hydrocephalus or cerebral palsy.

In some circumstances there are neurological indications for operative intervention. This does not necessarily imply that correction of the deformity is the prime aim but rather that the neurological signs are relieved, although the two often go together. Untreated, scoliotic deformities are not associated with spontaneous neurological symptoms and signs. The spinal cord is not therefore embarrassed in the lordoscolioses. Only when there is spinal dysraphism in one of its forms as an additional feature to the scoliosis can the spinal cord be tethered. In this respect the diastematomyelia in association with a congenital spine deformity is particularly important.³⁰ It is in association with kyphoses that most neurological problems arise.³¹ This is because the spinal cord is bowstrung behind the kyphosis and stretched over its apex. This may happen as a result of a congenital kyphosis or one associated with tumour or infection. It is extremely rare for it to be encountered in association with Scheuermann's disease. The management strategy is particularly important because as the spinal cord has been pressed on from the front it must be decompressed from the front, and thus laminectomy is not only futile but

positively harmful by way of removing essential posterior support.¹²

The underlying condition

Idiopathic scoliosis and kyphosis exist in children who are otherwise entirely normal. In other situations therefore the nature of the patient may become very important. Thus, while it would be very appropriate to correct the deformity of non-progressive congenital muscular dystrophy if it was threatening function, there is a widely held view that a similar deformity in association with Duchenne dystrophy with its second decade fatality should not be treated. On the other hand Marfan's syndrome, albeit with its cardiovascular complications, has a much longer survival and therefore the comparable deformity should be treated. With congenital spine deformities which are rigid and associated with a high incidence of neurological complications if not treated properly,³³ particular care must be exercised by way of preoperative investigation in order to minimize the neurological risk. In patients with neurofibromatosis those who have skeletal evidence of dystrophic bone change require all the support they can get in order to preserve the corrected situation and to prevent subsequent progression. Thus it is a general rule in such patients that spinal fusion should be performed both anteriorly and posteriorly. In patients with cerebral palsy the most severe spinal deformities are encountered in those whose intelligence is most severely impaired, and so it is that the very individual who may need corrective spinal surgery has the least ability to cooperate with the necessary rehabilitation programme. Moreover, in more than 50% of patients with cerebral palsy there is a significant sensory impairment in addition to motor dysfunction. And so it is that the underlying condition has a lot to do with management strategy and it would be quite erroneous to divorce the spine from the remainder of the patient's condition.

PREOPERATIVE CARE AND INVESTIGATIONS

Studies of adults with untreated scoliosis have shown increased morbidity and mortality from cardiopulmonary disease from which it has been inferred that patients with curves measuring more than 60° have evidence of pulmonary dysfunction.^{34,35} These inferences are incorrect and what really matters is the age of onset. Pulmonary par-

enchymal growth continues to the age of 8 years and only if there is a significant chest deformity (curve measuring 60°) by this time will cardiopulmonary function be significantly jeopardized.¹⁶ The great majority of scoliosis operative work concerns the late onset adolescent idiopathic case and for cosmetic reasons. Thus although it has been traditional to assess cardiopulmonary function preoperatively this must be regarded as a non-essential screening activity. Quite the opposite is the case with thoracic deformities of early onset in whom restrictive ventilatory defects and diminished gas transfer can be expected.

Preoperative cardiopulmonary assessment starts with a careful history with particular reference to dyspnoea which would indicate a marked reduction in lung volumes or a recurrent cough, asthma or previous cardiac or oesophageal surgery, all of which would predispose to postoperative respiratory problems. Examination is directed toward reduced chest expansion, excessive ventilatory effort, bronchospasm or secretions, all of which require further investigation. Congenital heart disease is not uncommon and has been estimated to occur in 5% of patients with scoliosis, but evidence of cyanosis is rare as most severe lesions will have been corrected in infancy. Nonetheless a praecordial murmur should be excluded and children with Marfan's syndrome may have aortic or mitral valve incompetence while prolapse of the mitral valve has been noted in Duchenne muscular dystrophy. A derotated anteroposterior view of the chest may be necessary so that the lung fields are not obscured by the scoliosis itself. However, right ventricular hypertrophy resulting from pulmonary hypertension is seldom seen under the age of 40. The electrocardiogram is influenced by the shape of the thorax and a tall P wave is of more significance than right axis deviation in predicting a raised pulmonary artery pressure as the shape of the thorax influences the direction of the QRS complex. Other changes such as a high QRS voltage and T wave inversion are also caused by the deformity.

In practice, a cardiopulmonary assessment is obtained on all patients preoperatively by an experienced chest physician. Forced vital capacity is measured and is considered normal if it is greater than 80%. Between 80% and 60% the restrictive defect is considered mild, if less than 60% moderate and if less than 40% severe. Patients in whom the vital capacity is less than 40% of normal should be prepared for assisted ventilation. Blood gas analysis is indicated when dyspnoea or signs of pulmonary hypertension are present. A Pao_2 of

less than 70 mmHg is indicative of pulmonary hypertension and this should be investigated with cardiac catheterization.

The intraoperative blood loss runs at between 15 and 25% of the blood volume and most series show a large postoperative loss. Accordingly it is wise to cross-match for half the blood volume. A moderate degree of hypotension with an absence of tachycardia provides excellent operating conditions in order to minimize operative loss and it is unnecessary to go to the extreme of profound hypotension (systolic blood pressure of 50–60 mmHg) which may jeopardize the vascularity of the spinal cord, particularly when it is under tension.¹⁷

The other chief area of preoperative concern is the status of the spinal cord. There is no risk of a neurological problem in straightforward idiopathic cases unless there is a spina bifida occulta in the lumbosacral region which may be a source of local cord tethering and for which preoperative CT myelography is necessary.¹⁸ Therefore an anteroposterior radiograph of the lumbosacral junction is essential. It is, however, with congenital bony deformities that the real risk of neurological insult arises during surgery. Because of the high prevalence rate of spinal dysraphism in association with these deformities preoperative CT myelography is essential. Anteroposterior plain films of the spine may show the site of the bony component of a diastematomyelia or the associated widening of the interpedicular distance. CT myelography confirms the nature of the lesion and indicates if the filum terminale is thickened (if the filum is visible it is thickened). Any source of spinal cord tethering can then be relieved in a preliminary first stage performed jointly with a neurosurgeon. The latter's counsel is particularly important as surgical removal may not reduce risks if the diastematomyelia is unusually extensive. One of the chief indications for surgery in kyphotic conditions is to relieve pressure on the spinal cord over the apex of the deformity, and thus preoperative CT myelography is important in defining the extent and severity of this compression.

OPERATIVE DETAILS

The spine is approached either posteriorly or anteriorly. Experience indicates that when the back and front of the same spine require to be approached it should be done not simultaneously but as a staged procedure with 2 or 3 weeks between stages.¹⁸ The majority of cases can, however, be dealt with by one posterior procedure.

Approaches

The posterior approach

The position of the patient is very important in order to reduce bleeding. The patient is placed face down with the legs sloping down at 30° to the horizontal (Figure 7.8). The abdomen must be free from pressure as failure to achieve this will lead to a much increased blood loss from inferior vena cava compression. Correct positioning cannot be achieved by the use of ordinary pillows and padded metal rests are necessary to support the pelvis and thorax. This raises the patient's abdomen some 5 cm above the table. The ECG is monitored and blood pressure measured either by a radial artery cannula or by an oscillotonometer.

The spine is approached through a mid-line longitudinal skin incision which should be only slightly curved in favour of the scoliosis. It is essential that the skin knife only incises about three-quarters of the depth of the skin. This is in order to avoid encountering subcutaneous blood vessels with a sharp instrument. The remainder of the incision is then performed with cutting diathermy which first incises the remaining depth of the skin and then the subcutaneous tissue down to, but not through, the deep fascia. In this way no appreciable bleeding occurs, and it is quite unnecessary to waste time injecting local anaesthetic and adrenaline in the belief that this will reduce

bleeding caused by sloppy surgery when all it will in fact do is to open tissue planes that are not normally encountered. The deep fascial layer is not opened until the whole depth of the subcutaneous space has been incised lest bleeding is caused which cannot be adequately controlled. The deep fascial layer is more clearly visualized if the subcutaneous tissue on each side of the midline is gently retracted manually to each side. The upper and lower thirds of the wound are now held apart by small self-retaining retractors. The spinous processes are now palpated through the deep fascial layer to identify their precise position and with cutting diathermy the deep fascial layer is now incised. If the line of this incision is kept precisely to the tips of the spinous processes the diathermy will be seen to divide an avascular 'white line' part of the deep fascia. The incision is continued with the diathermy until the tip of the spinous process or its cartilaginous apophysis is fully revealed.

The cartilaginous apophysis in the immature, or supraspinous ligament in the mature, is now incised sharply in the longitudinal axis of the vertebra through to underlying bone in order to ensure that the cut is subperiosteal. Into this incision is insinuated a Cobb periosteal elevator so that the periosteum is stripped from the posterior elements. By feeling with the instrument the periosteum is elevated out to the tip of the transverse process and immediately this subperiosteal space is packed with a gauze swab which provides haemostasis

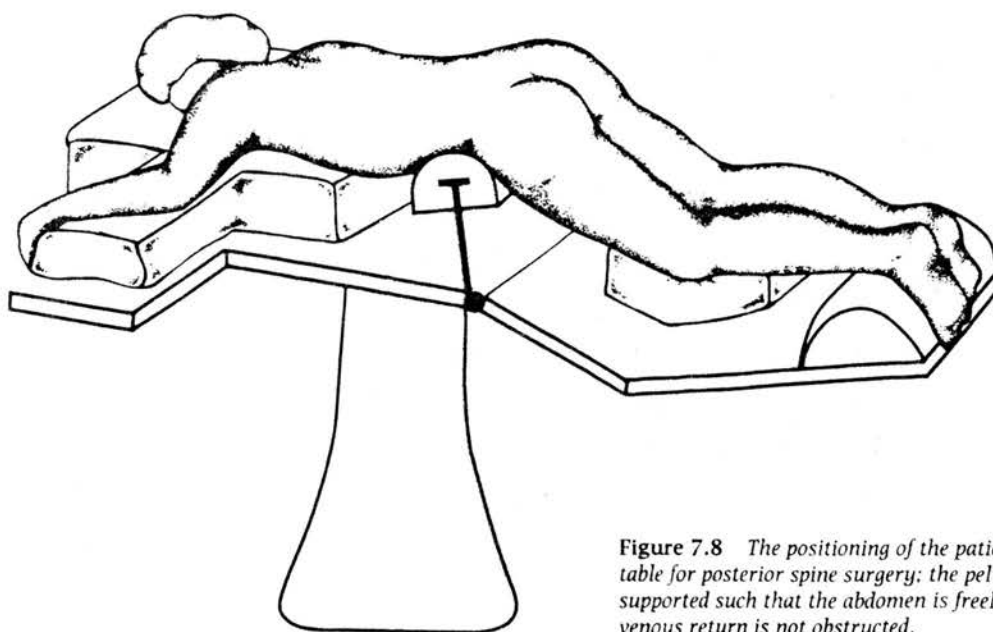


Figure 7.8 The positioning of the patient on the operating table for posterior spine surgery; the pelvis and chest are supported such that the abdomen is freely dependent and venous return is not obstructed.

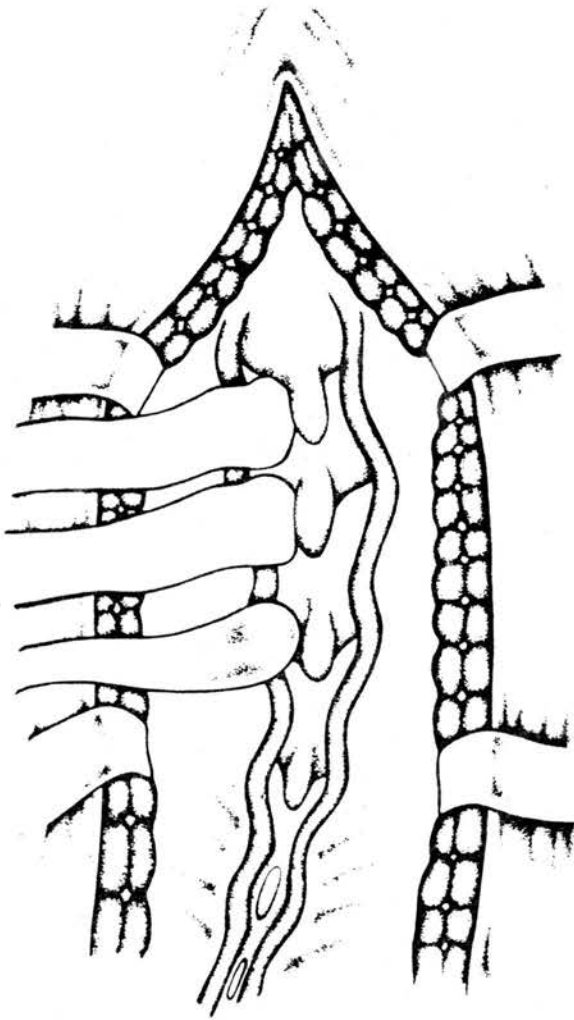


Figure 7.9 *The posterior approach to the spine. The cartilaginous caps of the posterior spinous processes are exposed in the midline. The caps are then incised longitudinally and split with a Cobb elevator carrying the plane of dissection subperiosteally. After exposure each level is packed with a gauze swab to provide haemostasis while the exposure is continued caudally.*

and increases exposure. Thus the spine is cleared of soft tissue subperiosteally at each level, starting proximally and working distally and clearing the concave aspect of the vertebrae in one sweep, before doing the same in a proximal-to-distal direction on the convex side (Figure 7.9). Thus it is that the surgeon clears the concave side and his assistant the convex side. When both sides have been so cleared there will be the tail of a sponge emerging

from the subperiosteal space on each side of each vertebra from top to bottom.

Phase 1 of the posterior approach is now complete. Starting proximally and working distally and beginning with the concave side two consecutive sponges are removed, the periosteum and attached musculature is retracted and two consecutive levels are further cleaned of soft tissue, using a combination of small Cobb and rongeur. These two levels are then packed with a sponge pushed well laterally, and then the next two spaces distally are dealt with similarly. When all levels on both sides have been dealt with in this manner phase 2 is complete. The sponges are now removed and the superficial self-retaining retractor at each end of the wound is passed deeply to retract the periosteum and musculature from the exposed spine at each end. The middle two quarters of the incision are retracted using bigger self-retaining retractors inserted over the smaller ones. The entire spinal deformity is now visualized from proximal to distal end and out to the tips of the transverse processes on each side. Any residual soft tissue (supraspinous, interspinous and intertransverse ligaments, facet joint capsules) is now removed until all the posterior aspect of the spine is completely denuded of soft tissue with the exception of the ligamentum flavum. During this stage of final cleaning segmental vessels at each interspace often require coagulation. Phase 3 of the approach is now complete and the spine is ready for instrumentation, or fusion, or both.

Anterior approach

The approach depends upon the site of the deformity and thus a thoracic, thoracoabdominal, or abdominal exposure may be required. The patient is placed in the lateral position, and if the operation is for a scoliosis the convexity of the curve is uppermost (Figure 7.10). If the spine is being exposed for a kyphosis then the side uppermost depends upon local anatomy (through the right side for most thoracic cases, and through the left side for most thoracoabdominal and abdominal cases).

Anterior approach to the thoracic spine. The approach is through the bed of the rib attached to the vertebra above the apex of the deformity so that there is no overhang preventing adequate access. This is performed through a standard lateral thoracotomy. The skin is incised sharply from midway between the medial border of the scapula and the spine posteriorly, two fingerbreadths below the inferior pole of the scapula, downwards and

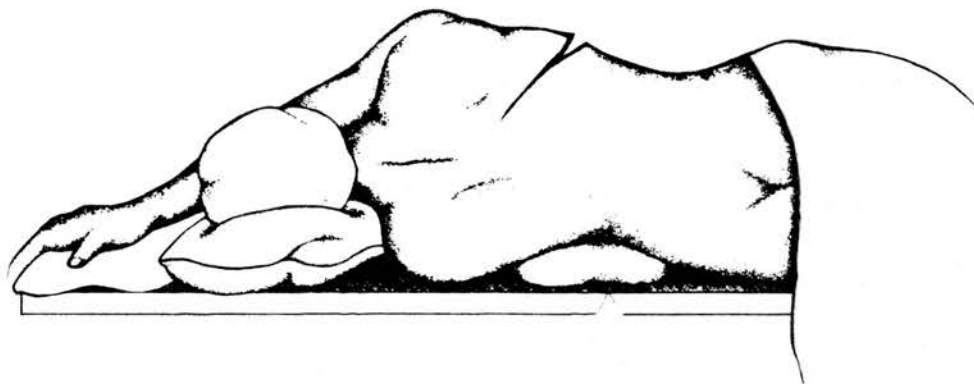


Figure 7.10 Positioning of the patient for anterior spine surgery; the patient is placed on the operating table in the lateral position with the side of curve convexity uppermost.

anteriorly as far as the anterior costal margin if necessary. The triangular space bordered by the lateral margin of trapezius and posterior margin of latissimus dorsi is located and entered with blunt dissection. The latissimus dorsi is elevated from the chest wall and divided by cutting diathermy as far anteriorly as the lateral border of pectoralis major, or further if more exposure is required.

The ribs are now exposed and the correct rib for excision selected. The periosteum over the external surface of the rib is divided by cutting diathermy and reflected from the rib itself. The rib is now excised by dividing it anteriorly with bone-cutting forceps and dissecting it from its vertebral attachments posteriorly. If access to the costovertebral joints is restricted, the rib can be divided at its neck and the posterior portion removed from inside the chest after the pleura is opened. The parietal pleura is then divided and the rib on each side retracted to expose the contents of the chest. If a double-lumen tube has been used then the lung on the operated side can be deflated to optimize exposure. The apex of the scoliosis projects towards the wound and important vascular structures fall into the concavity. By contrast, if a kyphosis is exposed, the spine is in the midline with a more normal relationship to other posterior mediastinal structures.

In order to expose the vertebrae themselves the parietal pleura is incised longitudinally over the centre of the vertebral bodies. At each level the segmental vessels are located as they course transversely over the vertebrae, and are divided between ligatures in the midline so as to preserve the integrity of the important longitudinal anastomotic vessels in the region of the intervertebral foraminae (Figure 7.11). The periosteum is then incised

longitudinally down the centre of the spine and reflected on each side as far as the intervertebral foraminae. Thus the vertebral bodies and intervertebral discs are exposed. If the whole length of the scoliotic deformity requires to be exposed anteriorly, as will be required to insert anterior instrumentation, then a second rib may require to be removed above or below to provide access of sufficient length.

Anterior approach to the thoracolumbar spine.

This approach is through the bed of the tenth rib so that the lower chest is exposed intrapleurally and the upper abdomen extraperitoneally. Should the deformity extend higher, then the ninth rib may require to be removed. The skin is incised along the line of the tenth rib which is removed subperiosteally as in the thoracotomy approach. The skin incision is continued anteriorly and inferiorly in the line of the tenth rib obliquely across the abdomen towards the midline (see Figure 7.11). The anterior abdominal wall muscles (external oblique, internal oblique, and transversus) are divided by cutting diathermy in the line of the skin incision. Care is taken not to transgress the peritoneum but if this occurs the defect should be repaired by suture at this stage. The peritoneum and its contents are then mobilized away from the abdominal wall by blunt finger and gauze dissection as far medially as the deformity dictates. The diaphragm is then divided between stay sutures along the line of the skin incision. The diaphragmatic crus on the convex side is dissected clear of the upper lumbar vertebral bodies to expose the entire thoracolumbar spine. The segmental vessels are again divided between ligatures in the midline

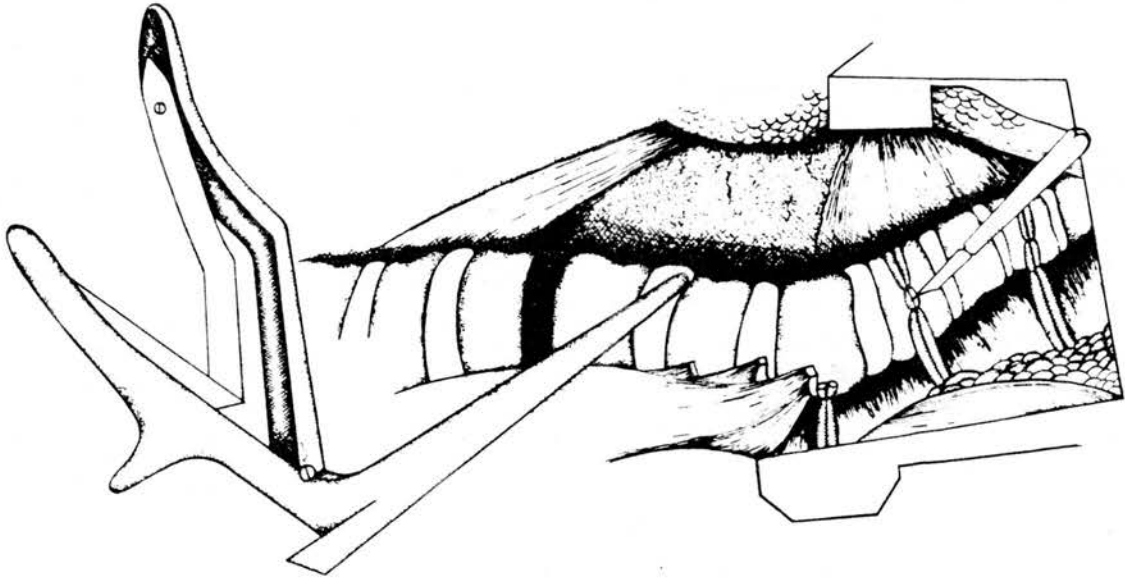


Figure 7.11 The anterior approach to the spine. Following exposure by a thoracotomy or thoracoabdominal incision, the segmental vessels are ligated and divided over the spine in the midline. Soft tissue and periosteal flaps are then raised with a Cobb elevator to expose the vertebral bodies and their intervening discs.

and the periosteum is incised longitudinally to expose the individual vertebrae and discs.

Anterior approach to the lumbar spine. For the great majority of requirements the extraperitoneal approach is used. The lumbar spine is approached through an oblique extended Rutherford-Morrison approach. Again the line of the skin incision should be placed above the apex of the deformity. Anterior abdominal muscles are again divided in the line of the skin incision and the peritoneum and its contents swept from the abdominal wall until the spine is exposed.

Rarely is it necessary to approach the front of the lumbosacral junction but for this purpose a transperitoneal approach is required through a standard lower paramedian incision. Before the anterior peritoneum is incised the retroperitoneal space can be insufflated so that the important autonomic nerve plexuses can be visualized. The peritoneum is incised longitudinally to the right of the midline. The median sacral vessels are divided between ligatures and diathermy is avoided. The common iliac vessels may require mobilization and superolateral retraction to expose the L5-S1 region from the front.

Instrumentation

Posterior instrumentation—Harrington

This comprises two sets of longitudinal rods and hooks—a distraction system on the concave side and a compression system on the convex side.

The distraction system. This consists of a longitudinal rod attached to the spine by hooks at the top and bottom of the curve. By increasing the length of the rod between the two hooks curve correction is achieved by distraction. The upper hook is seated differently according to site. Thus in the thoracic spine it is inserted under the inferior facet, while in the thoracolumbar or lumbar region, it is inserted under the lamina. The lower hook is seated under the lamina, unless instrumentation is taken down to the sacrum, in which case the lower hook rests over the sacral ala. A wide variety of hooks is available, being sharp or blunt and deep or shallow according to the local requirements (Figure 7.12). Thus the upper hook is sharp and has a right-angle flange so that it will bite into the pedicle below the facet and the flange will resist rotation. The lower hook is blunt because it seats under the lamina and is free to move until it comes under tension when the rod is

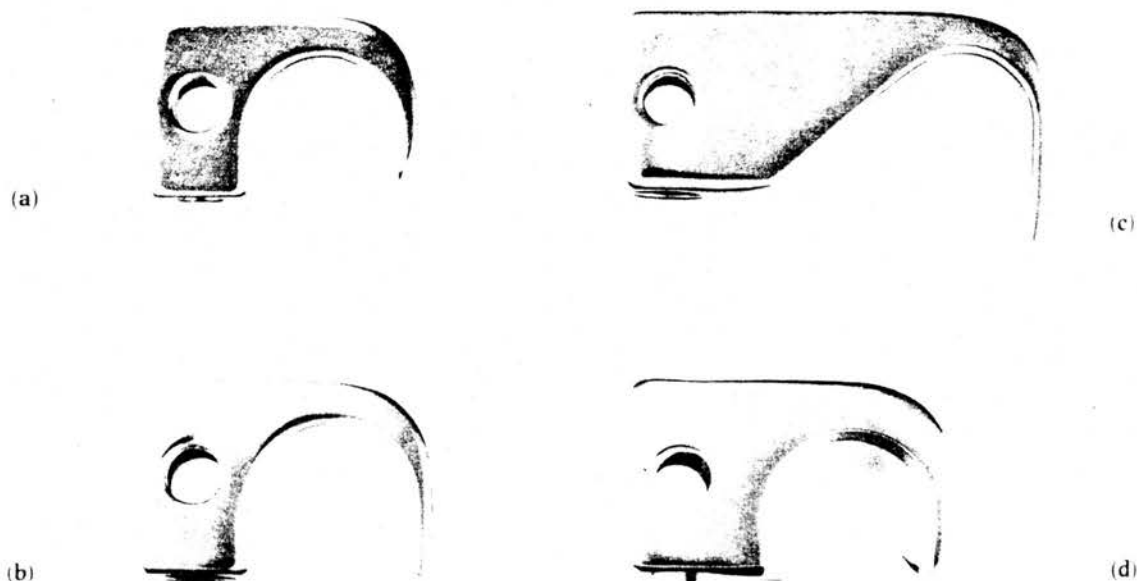


Figure 7.12 The different types of hooks involved in Harrington instrumentation: (a) the sharp upper hook, (b) the blunt lower hook, (c) the sacral ala hook, (d) the small sharp compression hook.

distracted. When the lower hook is seated over a deep bony buttress, such as the sacral ala, the special Leatherman deep sacral ala hooks are necessary.

For insertion of the upper hook the curved inferior margin of the lower facet of the neutral vertebra above is squared off using a small osteotome (Figure 7.13). Only a minimum amount of bone is removed so that as much of the facet as possible is left to sustain the hook. The sharp upper hook is then inserted using an introducer and is directed first in a posteroanterior direction before being hammered superiorly. This is so as the facet is not in danger of being split by the hook flange.

The ligamentum flavum on the concave side between the laminae of the lower neutral vertebra on the concave side and the one above is then removed using Colclough punches. The superior margin of the concave lamina of the neutral vertebra is then squared off with the Colclough punch taking the minimum amount of bone as is necessary to insert the lower hook. Insertion is facilitated by distracting the lower neutral vertebra and the one above using Backhouse towel-clips in the spinous processes. The distraction rod is now in-

serted and the ratchet end is pushed through the upper hook before the lower end engages the hole in the lower hook up to the collar. For most purposes the length of rod required is the distance between the upper and lower hook in the non-distracted situation plus four ratchets.

It will be appreciated from the introduction and patient selection that a standard Harrington distraction technique is seldom, if ever, used. In practice, a distraction rod is merely inserted so that segmental wires can be attached to it (Figures 7.5 and 7.14). For thoracic curves the rod is made physiologically kyphotic and in the lumbar region physiologically lordotic by the use of rod-benders. Rotation of these sagittally shaped rods is prevented by using a square-ended lower hook-rod relationship. For thoracolumbar curves a straight rod is used and thus a conventional lower hook-rod relationship is satisfactory. For added safety a second upper hook can be used and special upper hooks are available which allow some swivel without hook movement.

The compression system. This is an optional extra, used in uncomplicated idiopathic cases by only a

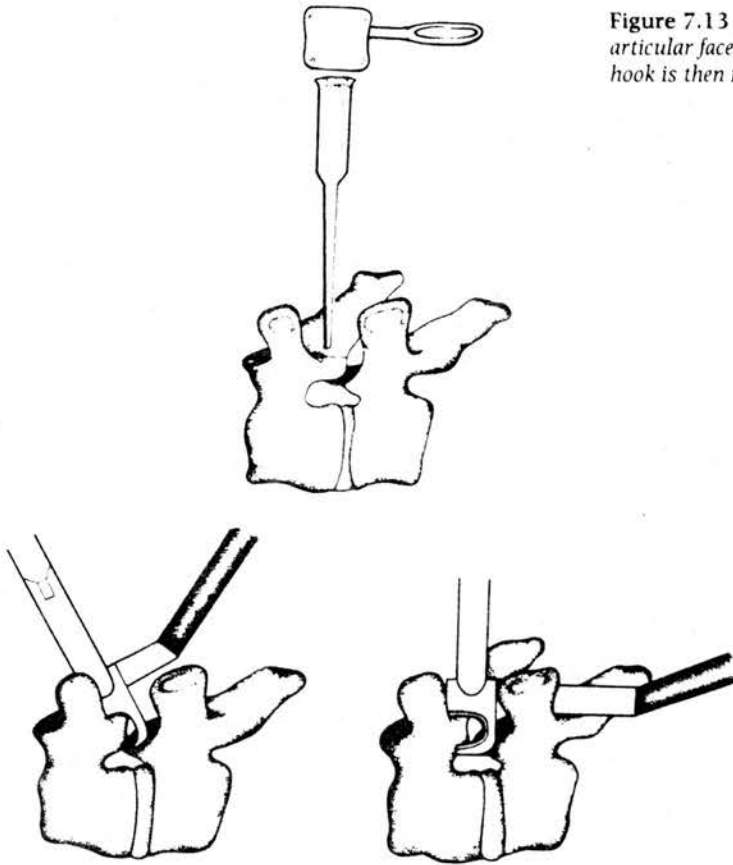


Figure 7.13 Method of upper hook insertion: the inferior articular facet is squared off with an osteotome and the hook is then inserted, first forwards and then upwards.

few surgeons, although mandatory for closure of the second stage of two-stage wedge resections. The rod is threaded and is of a smaller calibre than the distraction rod. It is fitted to the spine using multiple hooks seated over the transverse processes proximally and inserted into lateral masses or under laminae distally (Figure 7.15). There should preferably be three hooks pulling down above the curve apex and three hooks pulling up from below. The idea is that the system when tightened shortens the convex side, but it is doubtful whether it really does anything other than add stability but possibly increase the lordosis in the thoracic region because it is applied posterior to the vertebral bodies. There are compression systems available whereby the rod can be slotted into holes in the hooks which are first seated, but this can be as awkward to insert as the conventional system to be described.

A suitable length of compression rod is selected

according to the length of the convexity of the curve. Hook sites are now prepared. Three consecutive thoracic transverse processes above the curve apex are selected and using a joker (Legg retractor) and mallet, space is created for the flange of the compression hook above and anterior to each transverse process. It is most important that each compression hook can glide into place easily by being passed first posteroanteriorly and then inferiorly. A rotational insertion manoeuvre is not permitted as the definitive hook is to be threaded over the compression rod before insertion. A good place for the lower three compression hooks is into the lateral mass just medial to the facet joint in the thoracolumbar or lumbar regions. In this situation space may have to be made sharply using an osteotome. Otherwise the compression hook is seated under the most lateral aspect of the lamina after removal of the ligamentum flavum by Colclough punch or even better just lateral

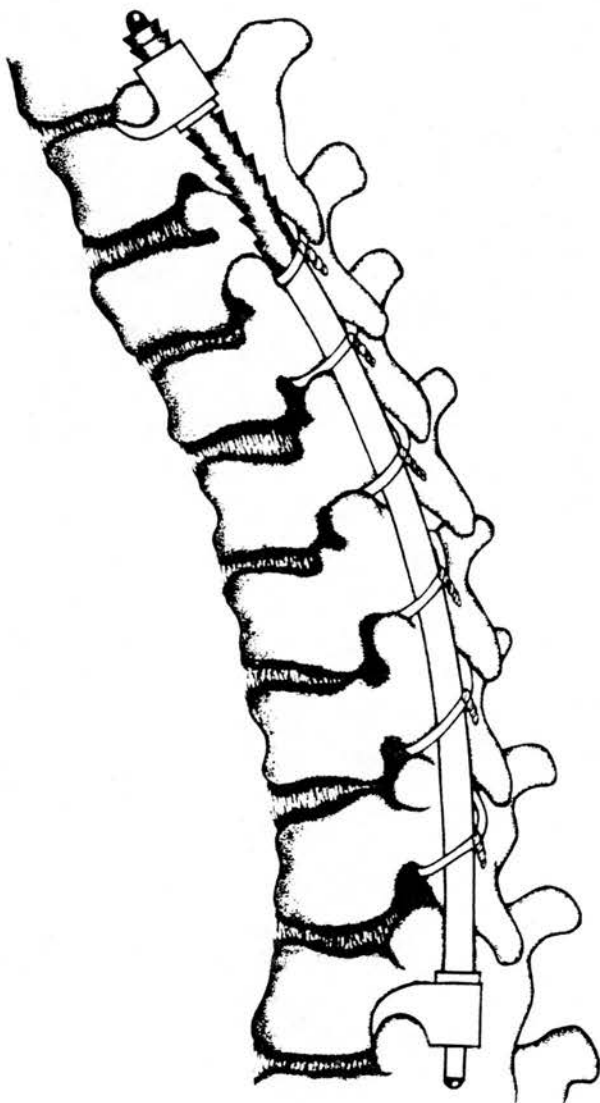


Figure 7.14 The spine instrumented with Harrington–Luque instrumentation. The vertebra are pulled back to a kyphotic Harrington distraction rod by sublaminar wires.

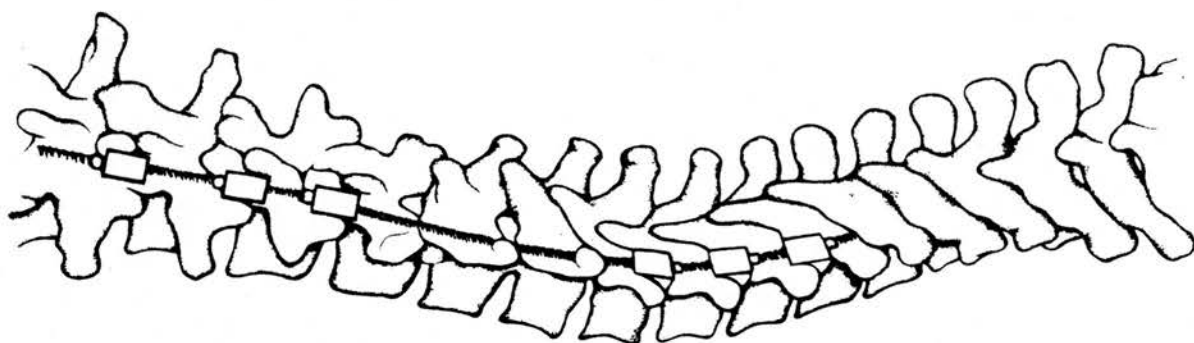


Figure 7.15 The Harrington compression system in place; compression hooks are placed round the transverse processes of the vertebral bodies and drawn together along the threaded compression rod.

to the ligamentum flavum. Again the compression hooks must be seen to glide into position freely without rotation.

These six compression hooks are then slipped over the compression rod and placed roughly in the right position on the rod and held there by nut chasing. The upper compression hooks are then inserted either one at a time or in combination, whichever seems appropriate, while the lower end of the compression rod is pulled inferiorly using the rod holder so that the upper compression hooks do not back out. Because of the scoliosis the lower end of the compression rod requires to be bent medially so that the lower three compression hooks can be seated while again tension is maintained on the lower end of the compression rod. Then by holding each hook with the hook-holder, and placing the rod-holder close by, each hook is tightened towards the curve apex by inserting a spreader between the two holding instruments and securing the tightened position by chasing the local nut. This is continued for each hook until maximum compression has been exerted.

If the distraction rod is now tightened this will loosen the compression system which requires to be tightened again, and thus distraction and compression systems are adjusted until the optimal correction is achieved. In order to restrain each end of the compression system from detaching itself from the spine and springing laterally they can be wired to the distraction rod. This should be performed at top and bottom only and not in the middle as approximation of the compression system and distraction rod will merely increase rotation as it is being applied behind the axis of rotation. For the same reason apical mini-compression systems¹⁹ also increase rotation and therefore are neither recommended nor described.

Sublaminar wiring techniques. For many years the posterior elements have been wired to Harrington distraction rods to aid stability and, of course, segmental wiring as a standard technique of posterior cervical spine arthrodesis. The Luque technique wires the laminae bilaterally to longitudinal rods which are not hooked into the spine. There are two ligamenta flava, one on the right and one on the left, and there is a plane of cleavage between them. This is developed bluntly using a Macdonald or Watson Cheyne dissector. The ligamentum flavum on each side of the midline is then excised using a Colclough punch and the contiguous margins of adjacent laminae are also removed to complete the 'laminotomy'. In the thoracic region the spinous process requires to be substantially re-

moved so that there is free access to the yellow ligament.

Stainless steel wire (50 cm of 1·10 mm diameter) is then bent double and passed under each lamina through adjacent laminotomies. To facilitate this passage the wires are bent to enable the folded apex of the wire to emerge through the second laminotomy (Figure 7.16). If these sublaminar wires are to be tightened against a Harrington distraction rod then laminotomies are only made on the concave side and the doubled wires are tightened around the distraction rod using a jet engine wire tightener or strong needle-holders (Figures 7.5 and 7.14). If the full bilateral sublaminar wiring and rodding technique of Luque is to be employed, particularly indicated for neuromuscular

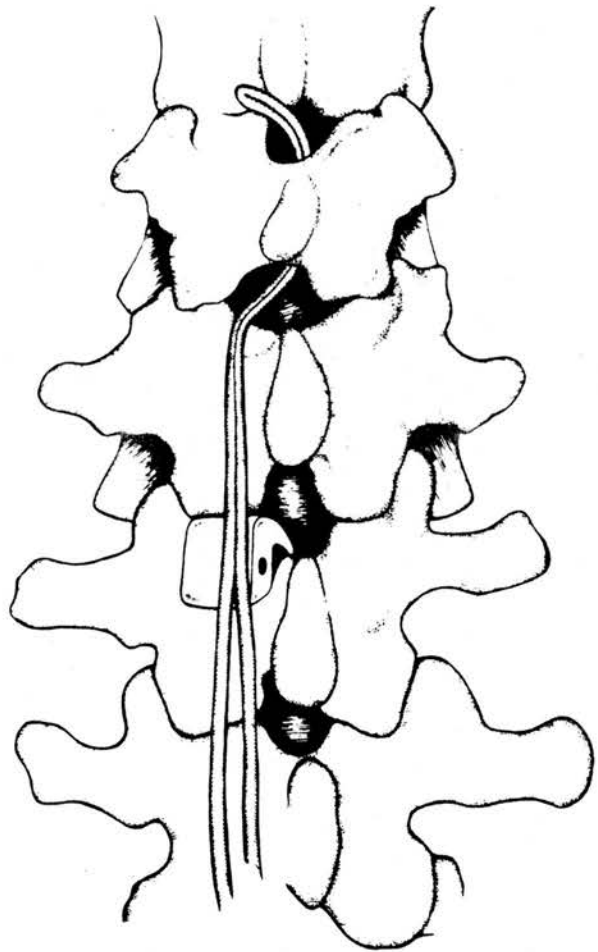


Figure 7.16 Double 20 SWG segmental wires are passed under the lamina from one laminotomy to the next.

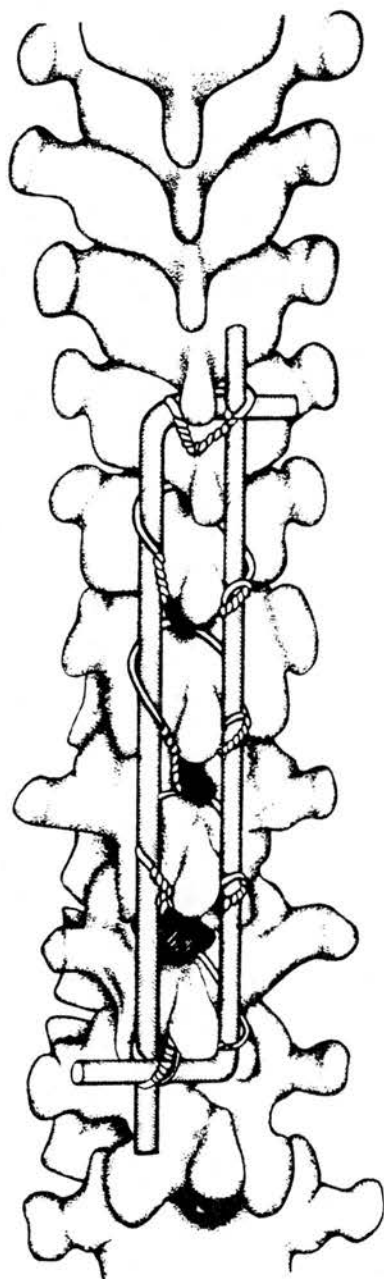


Figure 7.17 Posterior view of the spine showing the Luque instrumentation in place.

spinal deformities, then laminotomies are made bilaterally and the doubled wire is divided at its apex so that one strand passes to each of the convex and concave sides to be tightened round the longitudinal rods (Figures 7.5 and 7.17). These rods are 5 mm in diameter and are L-shaped with a short transverse limb. The transverse limb is at the

upper end of the L-rod on the convex side and the lower end on the concave side.

If a maximum side-bending or maximum traction radiograph shows that the spinous processes form roughly a straight line then the longitudinal part of the L-rod does not require to be bent. If these flexibility films show incomplete straightening then the rods must be bent to the estimated correction position before wire tightening. This is facilitated by drawing a line from the spinous process of the neutral vertebra above to the spinous process of the neutral vertebra below on the flexibility film and then measuring the distance to the side of this that the spinous process of the apical vertebra lies. Alternatively the amount of required bend can be templated in the operating room. For milder deformities and those in the thoracic region the convex rod is inserted first. The short transverse limb is secured to the lamina of the second vertebra above the curve and then wired to successive vertebrae down the curve using the lower end of the rod as a lever which, by drawing the rod to the midline, effects correction of the curve. Before the convex wires are fully tightened the concave rod is inserted and wired. For more severe curves or those in the lumbar region a concave rod is wired to the spine first. Manual pressure over the rotational prominence facilitates wire tightening. Before the concave wires are tightened the convex rod is inserted and the rods are approximated using a special clamp. This facilitates correction. When all the wires are tightened the free ends are trimmed and turned medially to avoid incorporation into the lateral fusion mass.

In neuromuscular conditions the L-rods require to be fixed to the sacrum and, the two short limbs being placed distally, these can be driven laterally into the posterior part of the ilium. In practice this is not necessary and the L-rods can be secured in the usual manner to the sacrum by wiring through sacral foraminae or through sacral ala according to local mechanical strength. Recently prefashioned rectangles of 5 mm rod (effectively two L-rods joined together) have been introduced which appear to be able to perform the functions of two L-rods and are fixed to the spine similarly, but with greater ease. In the spina bifida patient with deficient posterior elements the L-rods can be secured to the lateral bony buttress either using wires passed directly through bone or round the laterally splayed pedicles. If the L-rods or rectangle are used to stabilize the posterior aspect of the spine after anterior vertebral decompression, then correspondingly shorter lengths of L-rod and exposure are required.

Anterior instrumentation

Dwyer instrumentation. That there is always a lordosis in scoliosis led Dwyer to the concept of anteroconvex instrumentation associated with shortening achieved by intervertebral disc removal throughout the curve.¹¹ This technique is particularly suitable for thoracolumbar curves. After removal of discs and growth plates throughout the curve screws are inserted transversely across the centre of the vertebral bodies from convex to concave side (Figures 7.5 and 7.18). It is important that the end of the screw pierces the concave cortex and thus the appropriate length of screw must be selected following measurement. Each screw has a cannulated head in order to receive a cable of braided steel. When all the screws have been inserted the cable is threaded through the holes in the screw heads and by the use of a special cable tightening device adjacent screw heads are approximated, thus achieving a segmental correction, be-

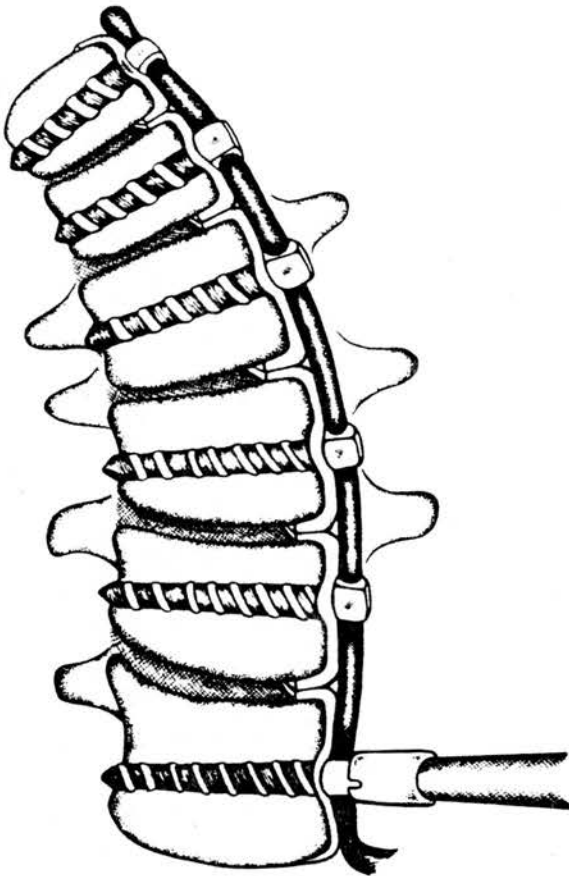


Figure 7.18 Anterior view of the spine showing the Dwyer instrumentation in place.

fore the screw head is crimped over the cable to secure the corrected position. This is repeated successively down the spine to the lower neutral vertebra.

This technique is particularly useful for achieving correction with stability in neuromuscular scolioses. Because of the firm attachment of L5 to S1 by the iliolumbar and lumbosacral ligaments, the pelvic obliquity which is a common accompaniment of a neuromuscular scoliosis can be favourably influenced by the Dwyer apparatus inserted only down to L5.¹¹ This is a great advantage as it is difficult to instrument to the sacrum. As the Dwyer cable is tightened the intervertebral disc spaces close. If closure is incomplete the gaps are packed with bone taken from either the rib excised or from the iliac crest or bone bank. If the vertebral body is particularly porotic the screw can be inserted well through the concave cortex and bolted.

Zielke instrumentation. The Dwyer instrumentation has recently been upgraded but essentially performs the same function (Figure 7.5). Instead of a cable there is a threaded rod similar to a Harrington compression rod and the VDS (ventral derotation spondylodesis) screws have slots in their heads for the insertion of the threaded rod. The use of special nuts prevents the compression rod from springing out of the slots. The VDS angle plates and washers are used in the terminal vertebrae in order to distribute the corrective force brought to the screw heads over a wider area. Instead of the screw heads being crimped over the cable as in the Dwyer system, tightening is effected by approximating screw heads by chasing nuts along the threaded rod. Before final tightening, derotation and lordosation (the latter not necessarily being desirable but unavoidable) are achieved using a special type of outrigger which employs the three-point principle being attached to the terminal vertebrae and the apical one.

Anterior instrumentation for kyphosis. Just as the front of the spine can be compressed using instrumentation so it can be distracted, and thus there are a variety of kyphosis distractors available.⁴⁰ They function on the same principle. Into vertebrae above and below the apex of the kyphosis a Dwyer or VDS type screw is inserted and the threaded rod which conjoins the screw heads is distracted until the desired amount of correction has been obtained (Figure 7.19). Although the distractor can be left in permanently it is more widely used as a temporary distraction device which provides room for an anterior strut graft, and when

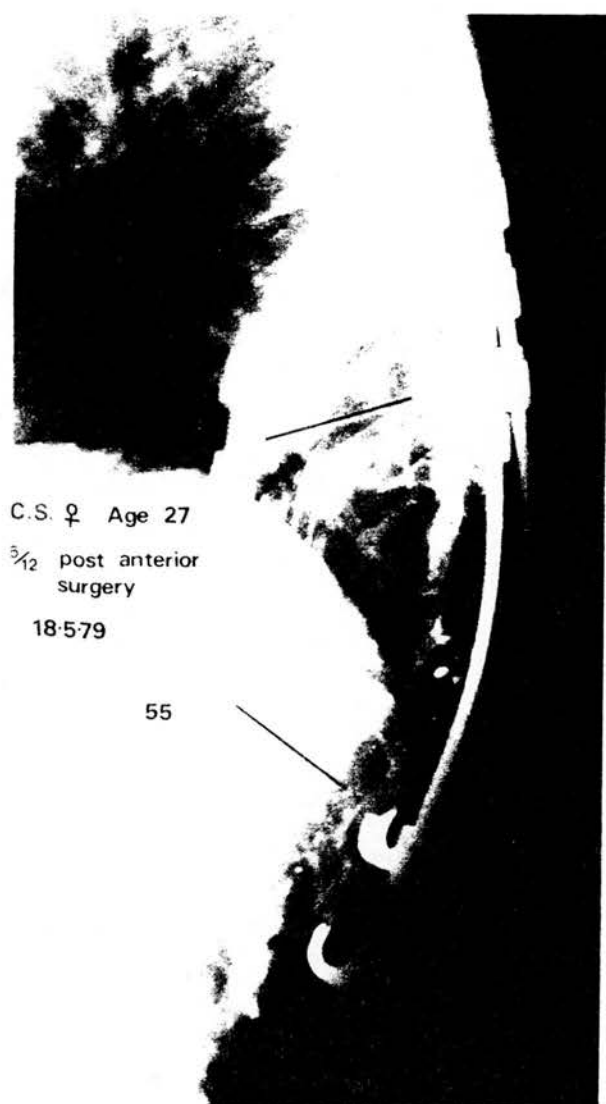


Figure 7.19 Lateral spine radiograph showing a kyphosis which has been corrected and strut grafted with the use of the kyphosis distractor. (Reproduced by kind permission of Mr A.D.H. Gardner, FRCS, and the Department of Medical Illustration, Basildon Hospital, Basildon.) (For a better quality picture see Publication 35, Figure 5.10.)

the distraction device is removed the graft will be under compression.

Other anterior surgical techniques

Anterior discectomy. This is a necessary part of the Zielke and Dwyer procedures but in practice is much more commonly employed for idiopathic scoliosis. For the curve measuring 70–90° a one-

stage posterior instrumentation strategy will not provide an adequate enough correction. Accordingly anterior discectomy throughout the curve provides room for the lordosis to be drawn through into a kyphosis (Figure 7.6). Often referred to as an anterior release, this is not so, as the front of the spine is too long and therefore under tension. Discectomy is straight forward and is facilitated by using an osteotome to develop the plane between vertebral bony end plate and epiphyseal plate. The intervening disc and growth plate material can then be removed by rongeur and curette until the posterior ligament is reached. The removal of five discs is sufficient for the typical curve. The definitive posterior instrumental procedure is then performed in the second stage.

Two-stage wedge resection. This is the most taxing but most rewarding of all the spinal procedures. It is specifically for the rigid deformity of any variety, the congenital deformity and the idiopathic case of more than 100°. Rather like the tibial osteotomy a wedge of bone is removed. The wedge is based on the convex side of the curve apex and provides a means of curve correction when the spine above and below is approximated. The wedge is removed in two stages (Figure 7.6), the front of the wedge from the front in the first stage and the back of the wedge from the back in the second stage. In the second stage the gap created by removal of the wedge is closed using a Harrington compression system on the convex side.

Anterior first stage

The anterior wedge to be removed is first scored out using an osteotome. It should aim to come to a point on the concave side at the site of a pedicle and facet joint. These structures on the concave side are not removed in the first stage because they are inaccessible but they provide a convenient hinge of stability between stages. They must of course be removed in the posterior second stage. A wedge of bone is then removed using a combination of osteotome and mallet, wood chisel, and double action rongeur. It is very important that as much bone be removed from around the spinal canal as possible before the spinal canal is breached. This is because irritating epidural bleeding can compromise subsequent bone removal. This particularly applies to bone concave to the spinal canal. If bone is removed in slivers there is no risk of driving the osteotome into the spinal canal. Bone softening or a change of colour as the posterior longitudinal ligament is reached signifies that the spinal canal is close by. It is a useful

landmark as attention can be directed to removing grossly bone from around the spinal canal.

When this bone has been removed the spinal canal is breached using a Macdonald or Watson Cheyne dissector. Bone and posterior ligament are then excised using Colclough punches. The wedge is completed when the pedicle, transverse process, and facet joint are removed from the convex side. Any bone bleeding is best stopped by the application of bone wax to the cancellous bone surfaces, while epidural bleeding is controlled by patés soaked in thrombin. At the end of the procedure the extradural fat or dura is covered by saline soaked gel foam.

Posterior second stage

In the second stage performed posteriorly the spine is approached in the standard fashion (Figures 7.8 and 7.9). Care is required in the subperiosteal exposure of the apical region lest preliminary anterior wedge resection has rendered the apex unduly mobile. The area of previous anterior vertebral body resection is located either radiographically on the operating table or by noticing the marks of the Colclough punch on the side of the posterior elements on the convexity. A standard laminectomy is then performed to complete the wedge posteriorly again coming to a point on the concave side. The loose pedicle, facet and transverse process must be removed from the concavity lest they infold during the closure process. The wedge is now closed using a compression system fitted to the convex side (Figure 7.15). Because the first stage has been accompanied by rib and transverse process removal both at the site of wedge resection and one level above the upper two compression hooks must be inserted two or three above the level of wedge resection. Only when the compression system is completely tightened and the wedge fully closed is a distraction rod added to the concave side and this is not distracted injudiciously as its function is one of stability rather than further correction.

Anterior decompression of a kyphosis. Anterior decompression of a kyphosis is indicated when there are signs of impending paraplegia. Accordingly cosmetic considerations do not pertain here and relief of neurological pressure is the aim of surgery. Therefore, while there are techniques available which can improve the degree of kyphosis, and thus it is assumed improve the neurological situation, such procedures cannot be guaranteed to produce the desired complete relief. The only certain method of achieving this is to totally remove the

offending obstruction. This implies anterior vertebral body resection at the apex so that the spinal cord is seen to be relieved of pressure. This is the most difficult aspect of anterior spinal surgery as the approach to the spine is not aided by the presence of a scoliosis. Moreover, the kyphosis is in the midline and it is exacting surgery to operate in its anterior concavity. Nevertheless this is essential for adequate decompression.

The apical vertebral body is removed by a combination of osteotome, rongeur, and Colclough punches. As in anterior wedge resection for scoliosis, as much bone surrounding the spinal canal is removed as possible before the spinal canal is breached. When this occurs the dura bulges forwards into its new-found freedom. Thus it is that as the aperture in the spinal canal is enlarged so this aperture is filled with bulging dura. As much bone is removed as is required to adequately decompress the spinal canal and this may be one vertebral body in moderate cases of kyphosis to three or more bodies in the most severe cases.

The resultant gap must be strutted by bone and, so that the strut graft of iliac corticocancellous bone is inserted under compression, an anterior kyphosis distractor is a useful adjunct as is removal of a disc or two above and below the area of vertebral resection to facilitate further reduction of the kyphosis (see Figures 7.6, 7.10, 7.11, 7.19). In addition, in order to add cancellous bone anterior to the spine for graft bedding purposes, the vertebral bodies at the apex of the curve can be osteotomized in the sagittal plane and hinged through 180° thus producing an osteoperiosteal flap.⁴¹ The anterior strut graft can lie against this flap so that ossification is more readily produced. Indeed by so doing it is generally unnecessary to consider the use of a vascularized strut of rib graft based on the intercostal vessels⁴² although this may be necessary when the bed is unsatisfactory as is occasionally encountered in radiation kyphoses. Any cancellous bone, provided it is healthy, removed during the process of spinal cord decompression is then added around the strut graft to facilitate bony fusion. If there is any doubt about the mechanical integrity of the situation following anterior decompression of a kyphosis it is always wise to add a second stage posterior spinal stabilization using the posterior segmental wiring technique with an added posterolateral spinal fusion if necessary (Figures 7.5, 7.14, 7.17).

For anterior decompression of the congenital kyphosis in myelomeningocele all the anterior soft tissue deforming forces must be divided (Figure 7.20). These are as follows—the crura of the dia-

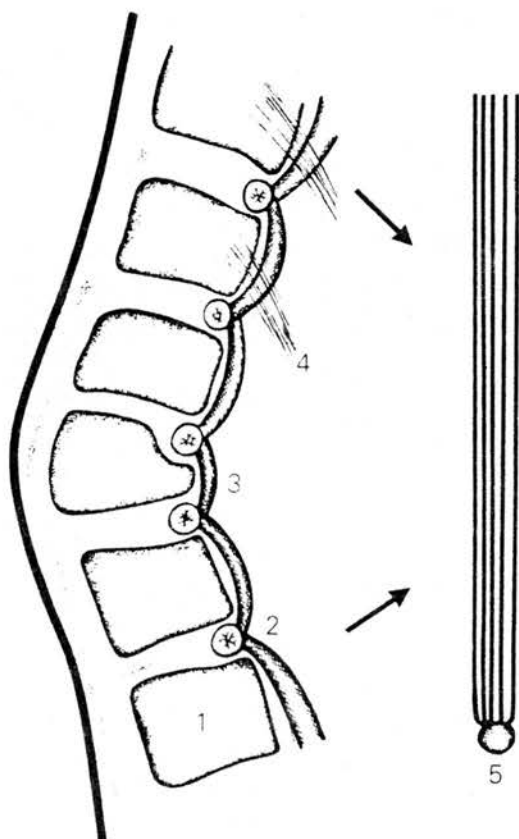


Figure 7.20 The deforming forces in the congenital kyphosis of myelomeningocele.

phragm, the attachments of the psoas muscle, the anterior longitudinal ligament, the annulae fibrosi and rest of the discs in the kyphosis. If the deformity still remains particularly rigid then anterior vertebral body resection at the apex is also necessary.

Fusion techniques

Posterior fusion techniques

The object of this exercise is to thoroughly decorticate the posterolateral aspect of the spine, and fusion should be from neutral vertebra above at least to neutral vertebra below at least. Therefore, meticulous removal of posterolateral soft tissues is necessary as a preliminary. The facet joints are first excised and this is performed using an osteotome which first removes the inferior facet of the vertebra above. This exposes the articular cartilage-covered superior facet of the vertebra

below and this cartilage is removed using a small gouge. Into the excised facet joint is inserted either a piece of local bone removed from the adjacent spinous process or a piece of bone graft material. If a single Harrington distraction rod is used then facet excision and fusion is performed on both sides, but if a compression system is used then the facets are only fused on the concave side lest interference with them on the convex side jeopardizes the integrity of the compression hooks purchased to bone.

The posterolateral surface of the spine is then decorticated. The spinous process, lamina, pars interarticularis and transverse process each have a sandwich configuration. There are two cortical layers enclosing a cancellous layer. Using a small osteotome these structures are split through the cancellous layer starting medially at the tip of the spinous process and working laterally (Figure

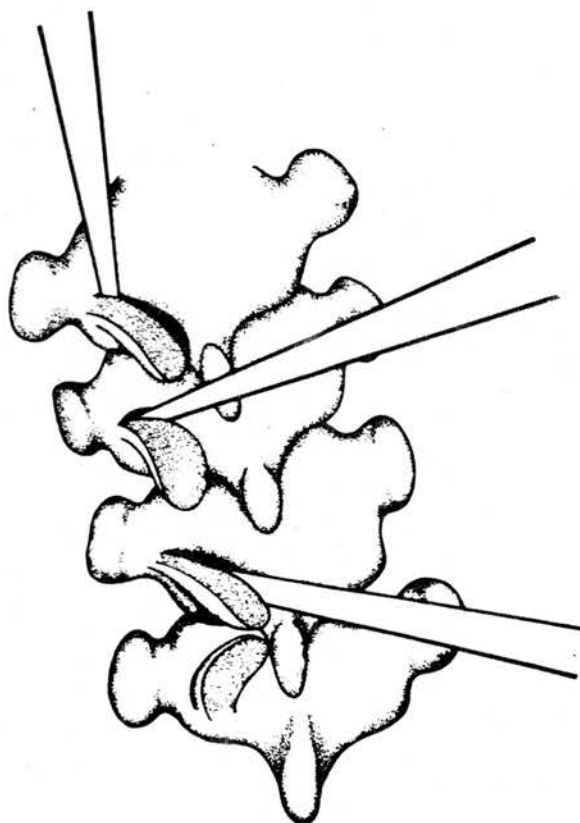


Figure 7.21 Method of performing a posterior spinal fusion. Flaps of local corticocancellous bone are raised using an osteotome and overlapped. This can be supplemented by the addition of local bone, cancellous bone graft from the posterior iliac crest or allogenic bank bone.

7.21). After about 1 cm the osteotome is turned upwards or downwards to produce a flap of bone still retaining an attachment to the spine and thus a vascular supply at its base. A fresh 1 cm flap is then started until the tip of the transverse process is reached. The flaps are turned over so that there is a cancellous surface between adjacent decorticated levels so providing a good bed for bone graft material.

If standard Harrington instrumentation is used in a thoracic spine then no additional bone graft material is required to be added for satisfactory fusion to occur. In the lumbar region, however, the spine needs all the graft it can obtain and thus a copious quantity should be added after decortication. However, with the combination of Harrington distraction rod and concave sublaminar wiring the laminae on the concave side are not decorticated and thus there is a reduction in the area available for fusion. In these circumstances, despite being a thoracic scoliosis, bone graft material should be added. Therefore the more instrumentation that has been inserted and the lower in the spine the curve, the more the need for bone graft material. This has traditionally been obtained in the nature of cancellous matchsticks from the iliac crest but allograft cancellous material from the bone bank is equally as good a fusion material. This is a very important consideration as it obviates the need to either extend the midline incision lower down to gain access to the posterior parts of the pelvis or make a separate oblique incision over the posterior part of the pelvis, an incision which heals notoriously badly. Additionally, a significant component of postoperative discomfort comes from the pelvis when this has been disrupted for the acquisition of bone graft material. Our practice therefore is to add cancellous allograft bone chips from the bone bank to all spinal fusions until the posterolateral aspect of the spine is covered with local and bank bone.

Anterior spinal fusion

The importance of anterior spinal fusion is becoming increasingly recognized particularly for early onset cases in whom suppression of anterior growth is desirable. The techniques of anterior fusion differ according to whether the deformity is a lordoscoliosis or a kyphosis.

In a lordoscoliosis the front of the spine is too long by definition and the only way in which this spine can be fused anteriorly is by removal of discs and growth plates until the raw cancellous surfaces of each vertebra are exposed (see Figure 7.6),

and then the resultant gap is filled with bone graft material unless it is to be compressed by anterior instrumentation. Because of failure to appreciate the three-dimensional nature of the typical scoliotic deformity (that is, lordoscoliosis) much emphasis is placed upon anterior strut grafting for scoliotic deformities. It is, of course, not possible to perform this procedure as the front of the spine is already too long. Thus anterior strut grafts can only strictly be applied to the front of kyphoses. What in effect is being meant by an anterior strut graft for a scoliosis is strutting the concave part of the lordoscoliotic deformity via an anterior approach. Thus if an anterior strut graft is performed for a scoliosis then the only structure separating the strut graft at the front from the Harrington distraction rod at the back are the ribs on the concave side. Therefore, the anterior strut graft is not anterior at all, but lies in the concavity of the deformity. Thus, effectively, the anterior strut graft could have been inserted from the back. The real importance of this is that when a scoliosis reaches a certain size, even in adulthood, it requires plenty of bone across its concavity to stop it collapsing further, though whether this is inserted from the front or back is irrelevant. If this so-called anterior strut graft procedure is performed, then the graft, a stout chunk of corticocancellous iliac crest bone, is in contact with the spine above and below the apex of the deformity, but is some distance from the spine at the apex and thus is relatively undernourished at this site and therefore weak. The gap between strut and apex should then be filled by raising an osteoperiosteal flap.⁴¹

The only situation where an anterior strut graft is really an anterior strut is at the front of a kyphosis. After decompression of the apex or mobilization of the kyphosis by multiple disc excision, the corrected position is maintained by the use of a stout corticocancellous strut of iliac crest bone keyed into the front of the spine (see Figure 7.19). It should lie close to the longitudinal axis of the spine and therefore may require to be keyed into the vertebra two or three above and below the apex of the kyphosis in severe cases. Each excised intervertebral disc space is filled with bone graft material and the gap between the apex of the kyphosis and the strut graft is filled with additional smaller corticocancellous struts, although of course, only one can be under compression. These struts should rest against an osteoperiosteal flap.

POSTOPERATIVE CARE

This really begins in the preoperative phase and extends through the operation into the postoperative phase if complications are to be avoided. Mortality and neurological morbidity are the real worries.³³ A cardiopulmonary assessment before surgery indicates the child at risk, and risks from this point of view should be discussed before a decision in favour of surgery is undertaken. The surgical techniques described and the use of experienced anaesthesia with moderate but not profound hypotension minimizes significant haemorrhage. In anterior wedge excision of the spine blood loss is occasionally considerable and this is due to epidural bleeding when these vessels are held open by the presence of an apical lordosis. With this type of bleeding local pressure cannot be applied because of the adjacency of the spinal cord and bleeding is best stopped using a combination of gel foam, sponges, and patés. As this bleeding is epidural it must be re-emphasized that the spinal canal should only be breached in anterior wedge resection after as much as possible of the extracanal bone has been removed, so that cessation of epidural bleeding is synchronous with the end of the procedure.

It is also important to realize that the same volume of blood that is lost during surgery is also lost during the first 24–48 hours thereafter, and so it is necessary to carefully monitor the filling of drainage bottles in relation to the vital signs of the patient and the rate of postoperative infusion. Perhaps the most important factor in reducing the amount of operative haemorrhage is to ensure that there is no pressure on the abdomen during the operation itself (Figure 7.8).

Postoperative chest complications are kept to an absolute minimum by the use of experienced anaesthesia. Low cuff pressure reduces damage to tracheal mucosa and cilia, and keeping the patient warm during the operation and the use of humidification reduce oxygen consumption and therefore minimize hypoxia. At the end of an anterior procedure both surgeon and anaesthetist inspect the lung to ensure that it is fully reflat in all areas, as large patches of collapse take a long time for the physiotherapist to reflate in the postoperative phase. The use of drains to maintain lung reflation and remove postoperative haemorrhage is, of course, mandatory.

The most important way of preventing postoperative chest complications is to ensure adequate pain relief. As the thoracotomy wound is being closed after anterior spine surgery intercostal

blocks or cryoprobe freezing of the intercostal nerves is performed routinely. Then as these wear off adequate opiates must be used to coincide with chest physiotherapy which should be used four to six times per day and at night if necessary. Physiotherapists use a Bird respirator to assist positive inflation. Repeat chest x-rays and clinical examination at regular intervals are important, with particular reference to the colour of the patient. All patients will be hypoxic to some degree whatever is done and the third postoperative day is notorious. Oxygen therapy (25–35% via a Ventimask or a Hudson mask with humidification) may be necessary. The circulating blood volume must be maintained as any reduction in the cardiac output makes pulmonary shunting worse. Meanwhile nursing has an important role to play in log-rolling to minimize chest stasis. Indeed the thoracotomy patient should have a personal nurse for the first 3 days. The combination of anaesthetic expertise, pain relief, maintenance of circulation, all obviate the need for postoperative ventilation and the otherwise healthy child should not need this following thoracotomy. If spontaneous respiration is difficult despite this then a period of time on a ventilator may be required.

The most alarming complication of surgery for spinal deformity is paralysis. Again, the preoperative investigatory phase has the most to offer in preventing this catastrophe. The congenital spine deformity and the spina bifida occulta all should alert the clinician to the need for a CT myelogram before surgery. Short of obvious iatrogenic damage to the contents of the spinal canal during surgery, it is inconceivable that a typical adolescent idiopathic scoliosis case should finish up with a neurological problem. Nonetheless, for this and every other procedure where spinal shape is changed, particularly under tension, the functional status of the spinal cord must be known at the time of the instrumental change in spinal shape. This can be performed in two ways. The wake-up test of Stagnara can be performed⁴³ and the nitrous oxide is switched off while the patient breathes pure oxygen. Within a minute or so anaesthesia is light enough for the patient to answer a verbal command to move his feet or toes. This is proof positive that there is a neurological communication between ear and feet but it has been described, although rarely, that neurological function can be lost some hours or even days after surgery. A more exact method of determining the function of the spinal cord is electrophysiologically during surgery and evoked electrical potentials are monitored across the site of surgery.⁴⁴ Any loss in the normal

response to the applied stimulus implies potential damage and the insulting force should be reduced or removed until a normal wave pattern is re-established. Electrophysiological spinal cord monitoring is still in a developmental stage, and may well transpire to be too sensitive, but there is sufficient evidence to indicate that it should be used routinely in cases where spinal shape is changed.

Wound infection is minimized by careful surgical technique, the use of suction drains and the use of antibiotics prophylactically when the spine is instrumented. However, there are certain patient groups who are particularly vulnerable to infection of whom the myelodysplastic is notorious. Such individuals should be placed on prophylactic antibiotics during and after all procedures.

The postoperative regime for patients with a spinal deformity used to be a long trial of bedrest or plaster immobilization even after a straightforward posterior approach. Indeed the patient often used to be in a plaster localizer before and during the operation.²⁴ Such times have, however, passed and after the great majority of the operative procedures described the patient is routinely mobilized towards the end of the second week. It is quite reasonable to allow up to 2 weeks of bedrest so that patient discomfort is minimized and the soft tissues can heal. Precisely what the patient should wear around the torso until the fusion is mature depends upon the procedure performed and the underlying condition. For the straightforward idiopathic deformity we favour an EDF (elongation-derotation-flexion) cast⁴⁵ made of polyurethane with zips down each side so that it can be removed

for showering purposes (Figure 7.22). It is important to not only immobilize the spine until the fusion is mature but to continue influencing the shape of the torso for as long as possible. It would be quite erroneous to imagine that the operative procedure was any more than a phase in the overall management of the patient; and if the deformity can be influenced by a cast postoperatively, such as is possible with an EDF cast, then this should be insisted upon. This is particularly important for thoracic deformities with their rib humps. For thoracolumbar or lumbar curves an underarm crutch-type brace (KSO—Kosair spinal orthosis)⁴⁶ is sufficient. If patient compliance is thought to be dubious, or if a two-stage wedge resection has been performed, or if there is any doubt about the security of the fixation, then a plaster of paris EDF cast should be fitted.

For neuromuscular spinal deformities a prolonged period of bedrest used to be necessary if anterior and posterior instrumentation and fusion of the Dwyer and Harrington variety had been performed, but with the advent of Luque instrumentation it has become clear that one of its greatest advantages is that it obviates the need for any form of postoperative support. Patients with these collapsing paralytic spines can be put back into their wheel chairs or ambulated forthwith. Nonetheless, there is strong circumstantial evidence that the rate of complications in the nature of instrumentation failure and pseudarthrosis is slightly reduced if some form of lightweight torso support such as a KSO is prescribed.

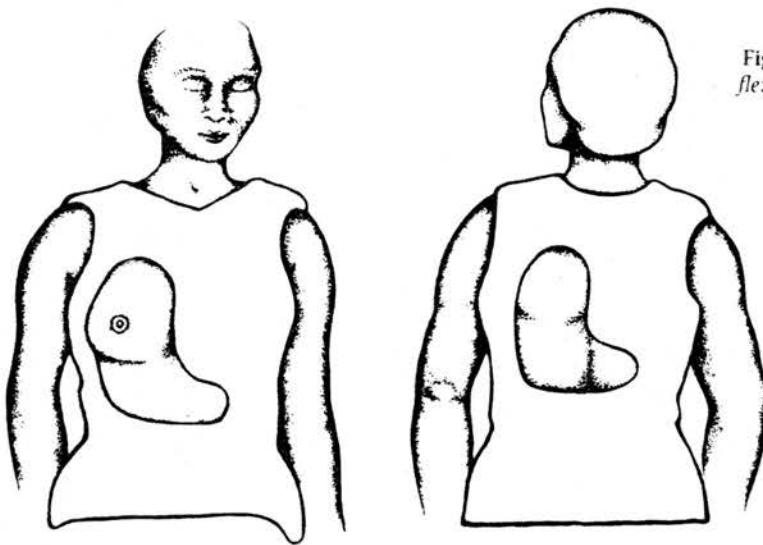


Figure 7.22 The EDF (elongation-derotation-flexion) postoperative cast.

OUTCOME

The results of plain Harrington instrumentation for scoliosis are not good⁷ and this procedure alone is therefore not recommended. Only of the order of a 50% correction of the coronal plane deformity was achievable with little or no improvement in the rotational prominence. While it can be argued that for small curves, around 40°, the result is reasonable it has to be said that there is no reason why the result in this sort of early flexible case should not be near perfect using newer systems. Moreover the younger the patient in whom a posterior fusion is performed the more is the danger that the fusion posteriorly will augment the pre-existing lordosis and so favour further tethering and rotational progression, often at a worse rate than before.^{2, 26-28} Thus it is that many of the undesirable effects of spinal deformity surgery are preventable. Certainly under no circumstances should a posterior fusion alone be performed on an early onset scoliosis. With the use of combined Harrington and concave Luque instrumentation all three components of the deformity can be corrected substantially.^{3, 10} The thoracic kyphosis can be restored, the coronal plane component and rotation improved by more than 50% each. Therefore there is much greater patient satisfaction with this type of approach and it must be remembered that this is the indication for surgical intervention in the great majority of cases.

Early attempts at producing a spinal fusion were associated with an unacceptably high prevalence rate of pseudarthrosis. As surgical techniques have improved, as the proportion of polio curves has diminished, and as newer instrumentation regimes have developed providing greater spinal stability, the rate of pseudarthrosis has fallen appreciably. Additionally, with the increasing use of allograft cancellous bone there is no need for a patient to be short of graft material. The pseudarthrosis rate for an uncomplicated idiopathic case must therefore be put at below the 1% level, although it is still of the order of 20% for the difficult myelodysplastic, and more than 20% for radiation-induced scoliosis where there is poor quality vascularity of local bone.

Proper insertion of the instrumentation should obviate hook or wire cutout and if the bone is unusually soft, as is encountered in conditions such as osteogenesis imperfecta or radiation scoliosis, then the metalwork can be bonded to the spine with methylmethacrylate cement. In these difficult cases with high rates of instrument and fusion failure the indications for the original operation cannot be overstressed. These are—in an attempt to prevent subsequent cardiopulmonary failure or to prevent sitting or walking functional impairment—and if surgery is addressed to these problems then there will be a higher percentage of successful results and a lower percentage of long-term complications. Allowing cosmetic considerations to enter into the surgical indications for these difficult cases is to invite long-term complications by necessarily increasing their prevalence rate. Therefore while patients with conditions such as Duchenne dystrophy can have their collapsing spines propped up from top to bottom with double rods and sublaminar wires, cardiopulmonary, instrumental and fusion complications are going to occur which would not be the case if the deformity were managed in an alternative way.

The high rate of complications following anterior instrumentation for scoliosis including loss of correction, pseudarthrosis, infection, metal failure and paralysis has led to recommendations that this form of instrumentation is rarely, if ever, indicated in uncomplicated idiopathic scoliosis. Indeed there is no reason why it should be used. However the high rate of complications with anterior instrumentation probably reflects the nature of the underlying condition being operated upon rather than the precise operative procedure.

When instrumentation is carried down into the lumbar region it must be bent to conform with a physiological lordosis as the postoperative straight lumbar spine is a severe cosmetic and functional problem. Wherever possible fusion down to L5 should be avoided as there is an association with derangements of the L5-S1 disc. Similarly fusion to the sacrum should be avoided except in paralytic cases associated with pelvic obliquity.

REFERENCES

- 1 Dickson RA (1983) Scoliosis in the community. *British Medical Journal* 286: 615-618.
- 2 Dickson RA, Lawton JO, Archer IA & Butt WP (1984) The pathogenesis of idiopathic scoliosis. *Journal of Bone and Joint Surgery* 66B: 8-15.
- 3 Dickson RA, Lawton JO & Butt WP (1984) The pathogenesis of idiopathic scoliosis. In Dickson RA & Bradford DS *Management of Spinal Deformities*, pp 1-37. London: Butterworths.
- 4 Dickson RA, Stamper P, Sharp AM & Harker P

- (1980) School screening for scoliosis: cohort study of clinical course. *British Medical Journal* 281: 265-267.
- 5 Sorenson KH (1964) *Scheuermann's Juvenile Kyphosis*. Copenhagen: Munksgaard.
- 6 Keiser RP & Shufflebarger HL (1976) The Milwaukee brace in idiopathic scoliosis. Evaluation of 123 completed cases. *Clinical Orthopaedics* 118: 19-24.
- 7 Aaro S, Dahlborn M (1982) The effect of Harrington instrumentation on the longitudinal axis rotation of the apical vertebra and on the spinal and rib-cage deformity in idiopathic scoliosis studied by computer tomography. *Spine* 7: 456-462.
- 8 Cardoso AM, Tajonar FA & Luque ED (1976) Osteotomias de columna nuevos conceptos. *Anales de Ortopedia y Traumatologia* 12: 105-113.
- 9 Allen BL (1984) Segmental instrumentation of the spine: indications, results and complications. In Dickson RA & Bradford DS (eds) *Management of Spinal Deformities*, pp 162-192. London: Butterworths.
- 10 Dickson RA, Lawton JO, Deacon P, Archer IA & Flood BM (1985) In Warner J & Mehta M (eds) *Scoliosis: Prevention*. New York: Praeger (in press).
- 11 Dwyer AF, Newton NC & Sherwood AA (1969) An anterior approach to scoliosis: a preliminary report. *Clinical Orthopaedics* 62: 192-202.
- 12 Griss P, Harms J & Zielke K (1984) Anterior instrumentation and fusion. In Dickson RA & Bradford DS (eds) *Management of Spinal Deformities*, pp 193-236. London: Butterworths.
- 13 O'Brien JP, Dwyer AP & Hodgson AR (1975) Paralytic pelvic obliquity: its prognosis and management and the development of a technique for full correction of the deformity. *Journal of Bone and Joint Surgery* 57A: 626-631.
- 14 Dickson RA & Leatherman KD (1978) Cotrel traction, exercises, casting in the treatment of idiopathic scoliosis. *Acta Orthopaedica Scandinavica* 49: 46-48.
- 15 Edgar MA, Chapman RH & Glasgow MMS (1982) Pre-operative correction in adolescent idiopathic scoliosis. *Journal of Bone and Joint Surgery* 64B: 530-535.
- 16 Ransford AO & Manning CWSF (1975) Complications of halo-pelvic distraction for scoliosis. *Journal of Bone and Joint Surgery* 57B: 131-137.
- 17 Leslie IJ, Dorgan JC, Bentley G & Galloway RW (1981) A prospective study of deep vein thrombosis of the leg in children on halo-femoral traction. *Journal of Bone and Joint Surgery* 63B: 168-170.
- 18 Leatherman KD & Dickson RA (1979) Two-stage corrective surgery for congenital deformities of the spine. *Journal of Bone and Joint Surgery* 61B: 324-328.
- 19 Stagnara P, Gonon G-P & Fauchet P (1984) Surgical treatment of idiopathic rigid lumbar scoliosis in the adult. In Dickson RA & Bradford DS (eds) *Management of Spinal Deformities*, pp 303-321. London: Butterworths.
- 20 Hodgson AR (1965) Correction of fixed spinal curves. *Journal of Bone and Joint Surgery* 47A: 1221-1227.
- 21 Dommissie G & Enslin TB (1970) Hodgson's circumferential osteotomy in the correction of spine deformity. *Journal of Bone and Joint Surgery* 52B: 778.
- 22 Houghton GR (1984) Cosmetic surgery for scoliosis. In Dickson RA & Bradford DS (eds) *Management of Spinal Deformities*, pp 237-251. London: Butterworths.
- 23 Hibbs RA (1911) An operation for progressive spinal deformities. *New York Medical Journal* 93: 1013-1016.
- 24 Goldstein LA (1966) Surgical management of scoliosis. *Journal of Bone and Joint Surgery* 48A: 167-196.
- 25 Moe JH (1972) Methods of correction and surgical techniques in scoliosis. *Orthopedic Clinics of North America* 3: 17-48.
- 26 Roaf R (1966) *Scoliosis*. Edinburgh and London: E. S. Livingstone.
- 27 Letts RM & Bobechko WP (1974) Fusion of the scoliotic spine in young children. *Clinical Orthopaedics* 101: 136-145.
- 28 MacMaster MJ & MacNicol MF (1979) The management of progressive infantile idiopathic scoliosis. *Journal of Bone and Joint Surgery* 61B: 36-42.
- 29 Harrington PR (1962) Treatment of scoliosis. Correction and internal fixation by spine instrumentation. *Journal of Bone and Joint Surgery* 44A: 591-610.
- 30 Till K (1969) Spinal dysraphism, a study of congenital malformations of the lower back. *Journal of Bone and Joint Surgery* 51B: 415-422.
- 31 Lonstein JE, Winter RB, Moe JH, Bradford DS, Chou SN & Pinto WC (1980) Neurologic deficits secondary to spinal deformity. *Spine* 5: 331-355.
- 32 Hall JE (1972) The anterior approach to spinal deformities. *Orthopedic Clinics of North America* 3: 81-98.
- 33 MacEwen DG, Bunnell WP & Sriram KA (1975) Acute neurological complications in the treatment of scoliosis. *Journal of Bone and Joint Surgery* 57A: 404-408.
- 34 Nachemson A (1968) A long-term follow-up study of non-treated scoliosis. *Acta Orthopaedica Scandinavica* 39: 466-476.
- 35 Collis DK & Ponseti IV (1969) Long-term follow-up of patients with idiopathic scoliosis not treated surgically. *Journal of Bone and Joint Surgery* 51A: 425-445.
- 36 Reid L (1971) Lung growth. In Zorab PA (ed) *Scoliosis and Growth. Proceedings of a Third Symposium*. Edinburgh: Churchill Livingstone.
- 37 Brodkey JS, Richards DE, Blasingame JP, Nulsen FE (1972) Reversible spinal cord trauma in cats: additive effects of direct pressure and ischaemia. *Journal of Neurosurgery* 37: 591-593.
- 38 James CCM & Lassman LP (1972) *Spinal Dysraphism, Spina Bifida Occulta*. London: Butterworths.
- 39 Cotrel Y (1978) Techniques nouvelles dans le trait-

- ment de la scoliose idiopathique. *International Orthopaedics* 1: 247-265.
- 40 Pinto WC, Avanzi O & Winter RB (1978) An anterior distractor for the intraoperative correction of angular kyphosis. *Spine* 3: 309-312.
 - 41 Stagnara P, Mauroy JC, Gonon GP & Campo-Paysaa A (1978) Scolioses cyphosantes de l'adulte et greffes antérieures. *International Orthopaedics* 2: 149-165.
 - 42 Rose GK, Owen R & Sanderson JM (1975) Transposition of rib with blood supply for the stabilisation of a spinal kyphos. *Journal of Bone and Joint Surgery* 57B: 112.
 - 43 Vauzelle C, Stagnara P & Jouvinroux P (1973) Functional monitoring of spinal cord activity during spinal surgery. *Clinical Orthopaedics* 93: 173-178.
 - 44 Tsuyama N, Tsuzuki N, Kurokawa T & Imai T (1978) Clinical application of spinal cord action potential measurement. *International Orthopaedics* 2: 39-46.
 - 45 Cotrel Y & Morel G (1964) La technique de L'EDF dans la correction des scolioses. *Revue de Chirurgie Orthopédique et Reparatrice de L'Appareil Moteur* 50: 59-75.
 - 46 Nash CL (1980) Current concepts review: scoliosis bracing. *Journal of Bone and Joint Surgery* 62A: 848-852.

IDIOPATHIC SCOLIOSIS : FOUNDATION FOR PHYSIOLOGICAL TREATMENT.

Dickson RA.

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This paper was based on a Hunterian lecture delivered in March 1984. It reviewed the concepts put forward concerning abnormalities of sagittal profile in the production of spinal deformity and reported on the effective correction of idiopathic thoracic scoliosis using segmental derotation in the first 25 patients.

Idiopathic scoliosis: foundation for physiological treatment*

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Key words: IDIOPATHIC; LORDOSCOLIOSIS; SAGITTAL SPINAL SHAPE; EXPERIMENTAL IDIOPATHIC SCOLIOSIS; PHYSIOLOGICAL TREATMENT

Summary

The three-dimensional nature of the idiopathic spinal deformity has been investigated in cadaveric specimens and patients with both idiopathic scoliosis and idiopathic kyphosis (Scheuermann's disease). In both scoliotic and kyphotic deformities the essential lesion lies in the sagittal plane with apical vertebral wedging. In idiopathic scoliosis there is an apical lordosis which being biomechanically unstable rotates to the side to produce a scoliotic deformity as a secondary component. In contradistinction the kyphotic wedging process of Scheuermann's disease is mechanically stable and any associated idiopathic type scoliosis occurs above and below the region of kyphosis. When an asymmetric lordosis is created in the growing New Zealand white rabbit, a progressive lordoscoliosis is readily produced and when the thoracic kyphosis is restored the scoliotic deformity shows evidence of regression and this forms the basis of physiological treatment. In 25 patients with idiopathic thoracic scoliosis the thoracic kyphosis has been restored and this leads to enhanced correction of the deformity in all three planes.

Introduction

Those of us who have had the honour and privilege to deliver a Hunterian lecture cannot but wonder at the extraordinarily catholic knowledge of surgical science that John Hunter possessed. Moreover, this knowledge did not only include surgery in general but encompassed the whole of the musculo-skeletal system from the structure and function of articular cartilage on the one hand to the complexities of three dimensional spinal deformities on the other, interestingly, it was not just the more obvious severe deformity which attracted Hunter's attention, of which there is a classic example in the Hunterian Museum, but also the mild and seemingly innocuous deformity present in the thoracolumbar spine of the 'surprising Irish giant' Charles O'Byrne, clearly discernible at the entrance of that museum, and in at least 15% of normal schoolchildren (1).

Somerville, one of the leading orthopaedic intellectuals of his generation, adopted the Hunterian approach of challenging conceptual assumptions with surgical scientific methods and thereby contributed greatly to our understanding of the nature of three dimensional spinal

deformities as well as providing a strong stimulus to this research programme (2).

The deformity of idiopathic scoliosis accounts for more than 95% of spinal deformities detected in the community and more than two thirds of patients attending scoliosis clinics. Unfortunately, little progress has been made in the last fifty years towards characterising this so common deformity and treating it effectively. Orthotic devices, thought at their inception and over the next thirty years to be able to attenuate the progression of the deformity (3), have recently been shown not to alter the natural history of the condition (4) and conventional posterior surgical techniques have been shown to provide a limited correction of the deformity in only one plane while leaving the rotational hump, with which every patient presents, essentially unchanged (5). In terms of our powers of correctability we have advanced little from the days of our scoliosis surgical father figures at the beginning of this century (6). Furthermore, comprehension of the nature of this three dimensional deformity has not been helped by the use of anatomically incorrect terminology. Although the time allocated for undergraduate orthopaedic teaching still sadly lags behind clinical reality, there can be few medical practitioners who have not encountered the word 'kypho scoliosis', a condition which, as will be seen, cannot and does not exist.

The aetiology of idiopathic scoliosis is unknown but, because a rotational spinal deformity is common in neuromuscular conditions, most pathogenetic work has sought to elucidate a disorder of nerve or muscle (7). No primary nerve or muscle lesion has thus far been detected and such changes as have been described are considered to be secondary to the presence of a spinal deformity (8). Clearly, if by x-ray screening a majority of children can be shown to have a measurable spinal deformity, 10% with curves measuring more than 5°, 2% with curves measuring more than 10°, and 1% with curves more than 15°, then a neuromuscular condition of epidemic proportions is being postulated. In addition, if a primary neuromuscular factor is present, it ought to be so in children with straight backs in whom the noxious agent has not yet produced a detectable deformity, unless its mode of action is to suddenly produce an acute scoliosis in every case, which is not a feature of any known neuromuscular condition.

Moreover, children with idiopathic scoliosis are otherwise entirely healthy normal individuals. Even if a specific agent were detectable, it would still put us at

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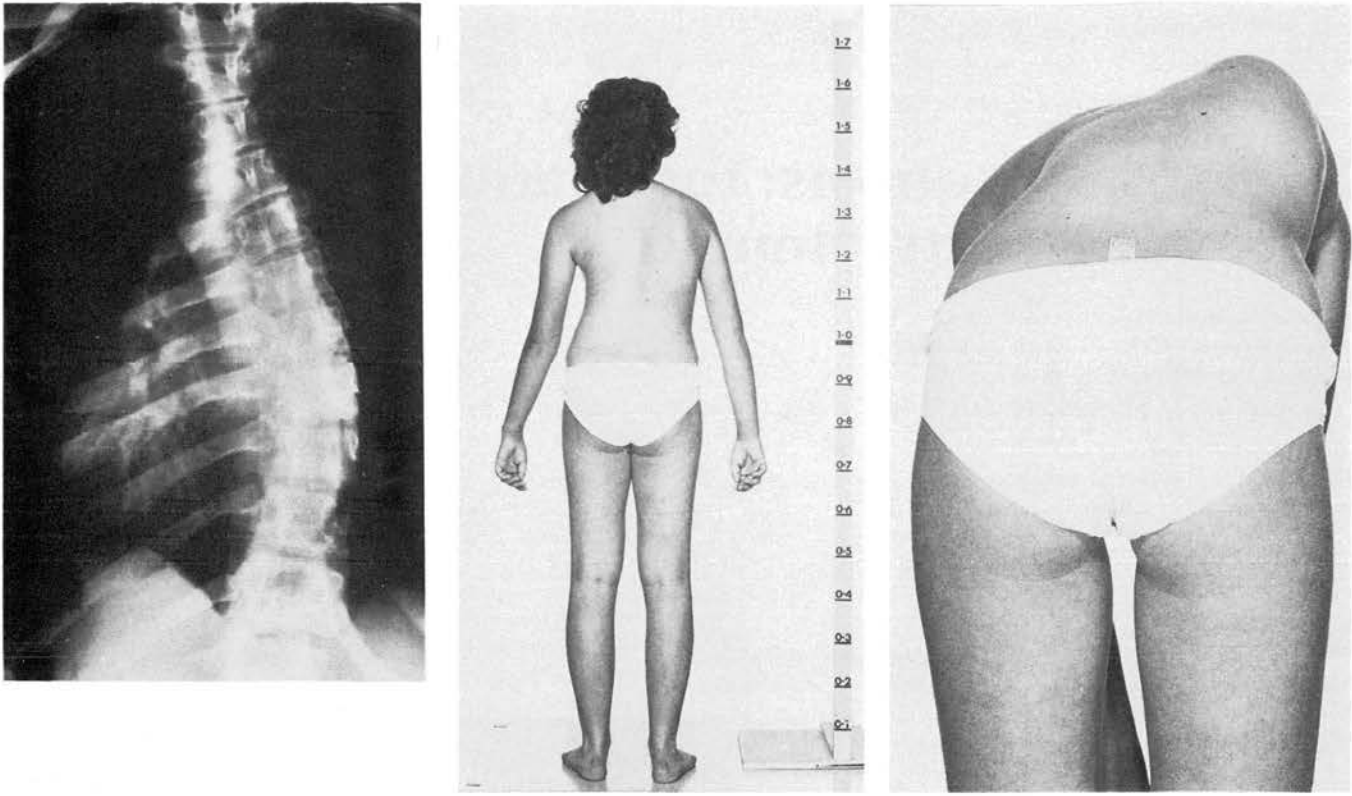


FIG. 1 (a) posterior-anterior (PA) X-ray of a patient with idiopathic scoliosis. The posterior elements are rotated into the curve concavity while the bodies are rotated into the curve convexity: the deformity must be lordotic throughout. (b) A patient with an idiopathic thoracic scoliosis standing. The deformity is less marked. (c) The same patient leaning forward. The rotational prominence is maximised on forward flexion.

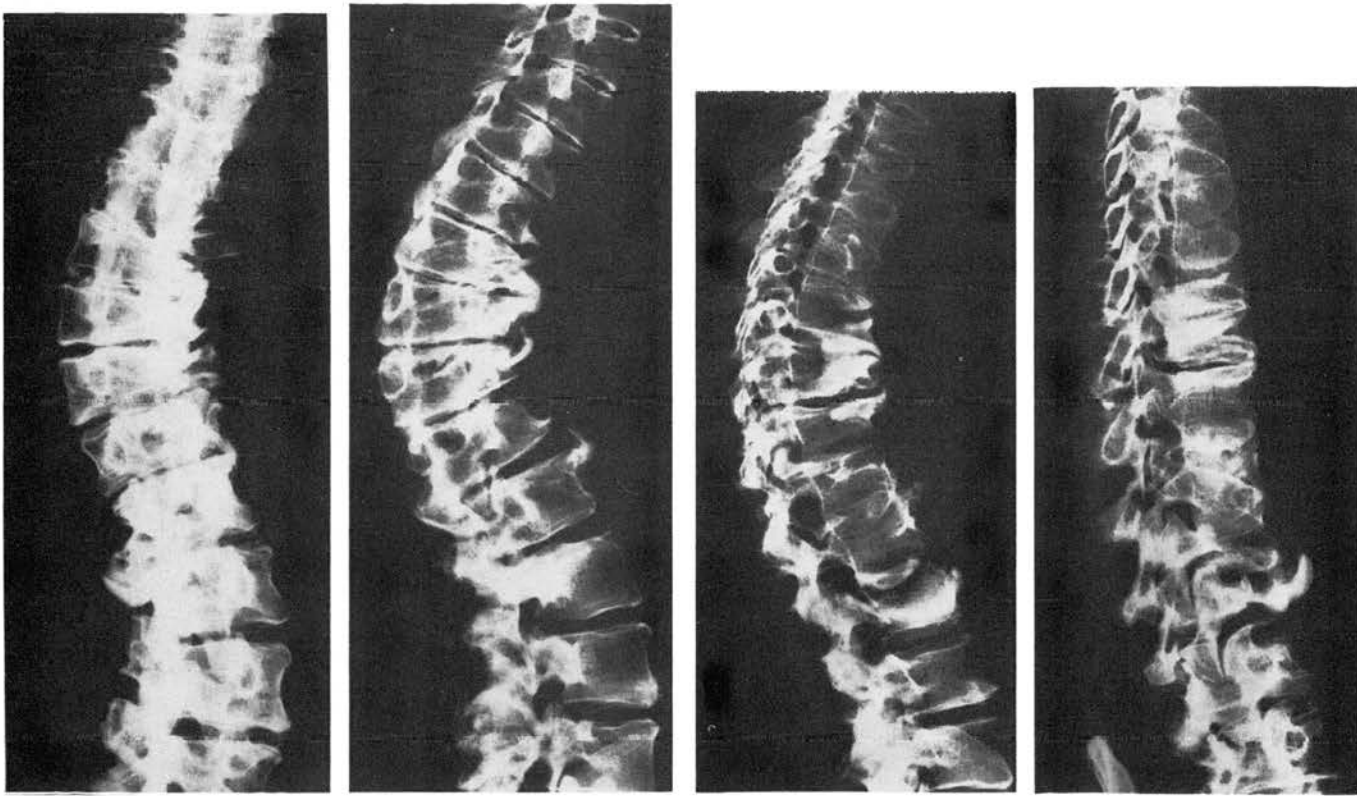


FIG. 2 Four radiographic views of a museum specimen of idiopathic scoliosis. (a) PA view of specimen; showing PA oblique view of deformity. (b) PA view of deformity; the scoliosis is now maximal. (c) Lateral view of specimen; showing lateral oblique view of deformity and the illusion of a kyphosis. (d) Lateral view of deformity; showing the apical lordosis.

a considerable distance from understanding the three dimensional spinal configuration produced, and its behaviour. Accordingly it appears more logical to start with an analysis of the deformity and to work from there.

Patients and methods

SIMPLE CLINICAL OBSERVATIONS

Idiopathic scoliosis can be defined as a lateral curvature of the spine with rotation and, interestingly, much about the nature of the deformity and its mechanical behaviour can be discerned from a single postero-anterior radiograph of the patient and inspection of the patient standing and leaning forward, (Fig. 1). In this typical thoracic idiopathic scoliosis, as with a similar deformity in any other site, the spinous processes are rotated towards the curve concavity while the vertebral bodies rotate towards the curve convexity. In the thoracic spine there is normally a smooth kyphosis (round back) but if a kyphosis rotates then the direction of rotation will be the reverse, with the spinous processes directed towards the curve convexity. It can therefore be said with some certainty that a thoracic lordosis is an integral part of the three dimensional deformity. This is verified by the elementary geometrical observation that if the spinous processes are directed towards the curve concavity then a line joining them from the top to the bottom of the curvature will describe a shorter distance than the vertebral bodies, which are directed towards the convexity. If the line of the anteriorly situated vertebral bodies is longer than the posteriorly situated spinous processes then the whole deformity must be lordotic. In the lumbar region of course a lordosis is already present as a normal sagittal curvature.

In the erect position the rotational prominence on the convexity of the scoliosis is very much less obvious than in the forward bending position (Fig. 1), indicating that a mechanical effect has been produced accentuating the rib hump with spinal flexion. In flexion the lordotic spine is under increasing compression such that it cannot be accommodated without rotating to the side. As the deformity is one of lateral curvature with rotation, then the PA radiograph of the patient, the standard view obtained for assessment and measurement purposes, is a PA view of everything except the deformity in question, of which it is a random oblique view according to the amount of vertebral rotation, and so of course is a lateral projection of the patient and not the deformity. In order to take true planar views to analyse the deformity either the patient or the beam must be rotated according to the amount of vertebral rotation present.

CADAVERIC ANALYSIS

To establish the true planar nature of the deformity radiologically without involving ethical considerations, attention was directed to museum specimens of idiopathic scoliosis (9). Unfortunately the bulk of Hunter's superb collection of scoliotic spines was destroyed by fire and the curator of the museum of our sister college in Edinburgh (Professor D C Meekie) kindly gave permission for their specimens to be studied. Each specimen was rotated through 180° and radiographed at 10° increments of rotation. Anterior and posterior vertebral body heights were also measured throughout the spine. The specimens included varying degrees of curve severity but a constant pattern emerged (Fig. 2). As the specimen was rotated from the PA position of specimen or patient, the size of the lateral curvature increased to a maximum, at which position the apex of the curve was truly PA. These observations demonstrated that the PA view of the specimen or patient underestimates the true magnitude of the deformity by 40% on average.

As the specimens were rotated further, so the size of

the lateral curvature diminished and at 90° of rotation round from the true PA projection a true lateral of the curve apex was obtained, which demonstrated an average lordosis of 14°. By contrast lateral projections of the specimen or patient give the spurious impression of kyphosis measuring on average more than 40°. Thus, not only did the PA and lateral projections of the specimen or patient provide quite erroneous information concerning the true planar nature of the deformity, but were nothing more than oblique views of the same scoliotic deformity. These views at different increments of rotation also enable all the vertebrae within the curve to be identified as regards their true rotational position in space. Morphometric measurements confirmed that all vertebrae within the curve were truly lordotic, anterior vertebral height being greater than posterior.

ANALYSIS OF PATIENTS WITH IDIOPATHIC SCOLIOSIS

One hundred and fifty patients with idiopathic thoracic scoliosis have now been analysed with reference to true

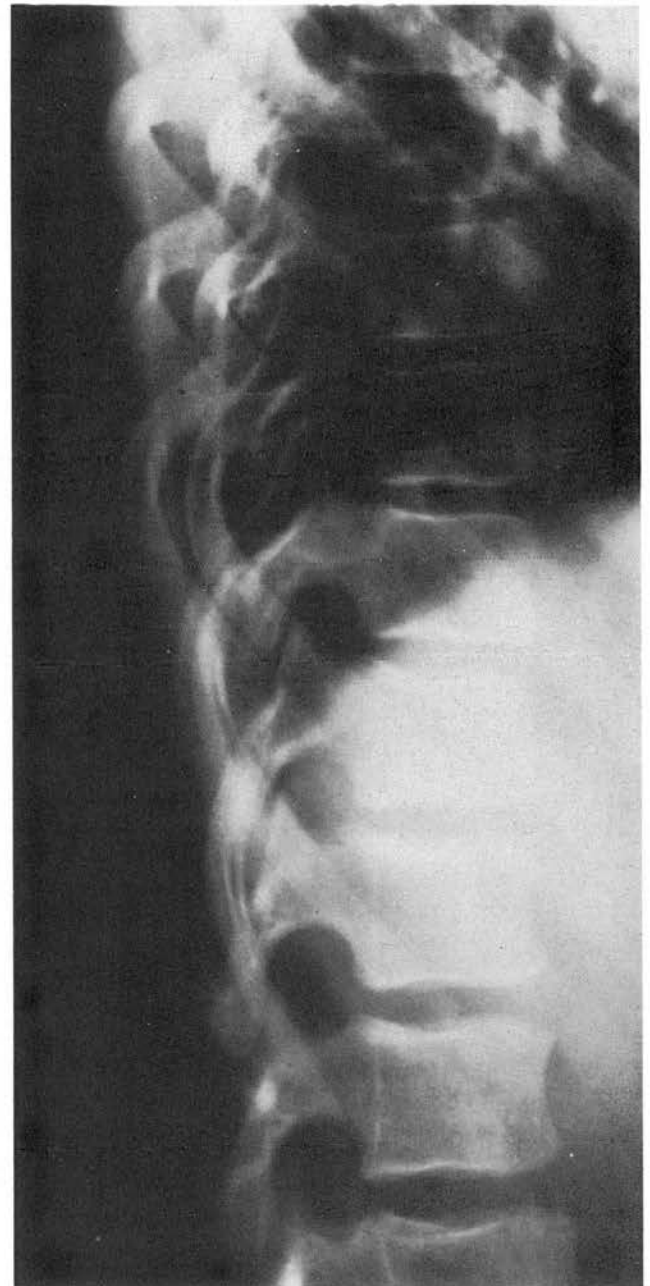


FIG. 3 True lateral radiograph of the apex of an idiopathic thoracic scoliosis showing the apical lordosis.

planar views of the apex of the deformity. Their mean age was 14 years and the female to male sex ratio was 7:1. True lateral radiographs of the apex of the deformity were obtained in all cases by fluoroscopy, computerised axial tomography or geometrical measurement (10). An apical lordosis was present in all cases (Fig. 3) affecting modally the apical three vertebrae, and the angle of lordosis, which range from 4° to 18° , was significantly correlated to PA curve size ($P < 0.001$), the bigger the lordosis then the bigger the associated scoliosis deformity. The position of the lordotic apex correlated significantly with the apex of the scoliosis ($P < 0.001$), with the ninth thoracic vertebra as the mode. Computerised axial tomography demonstrated that the apical vertebral bodies were least rotated one to another and that maximal intervertebral rotation occurred in the segments well above and below the apex, and this was strictly in the nature of derotation bringing the head and pelvis neutral.

ANALYSIS OF PATIENTS WITH SCHEUERMANN'S KYPHOSIS

The similarity in sagittal vertebral wedging between the lordosis of idiopathic scoliosis and the kyphosis of Scheuermann's disease prompted an analysis of 30 consecutive cases of the latter. PA and lateral views of the thoracic and lumbar spines of these patients were obtained and all had the diagnostic criteria of Scheuermann's kyphosis with mean vertebral body wedging of 8.5° extending over four consecutive segments. Twenty one of these patients had an associated scoliosis and in five cases these lateral curvatures were in the region of the Scheuermann's kyphosis with the vertebral bodies either not rotated or rotated such that the posterior elements were directed towards the curve convexity. By contrast 60% of these lateral curvatures were true lordoscolioses with posterior element rotation towards the curve concavity. These lordoscoliotic deformities were either above or below the Scheuermann's kyphosis in the region of the compensatory lordosis which had rotated to

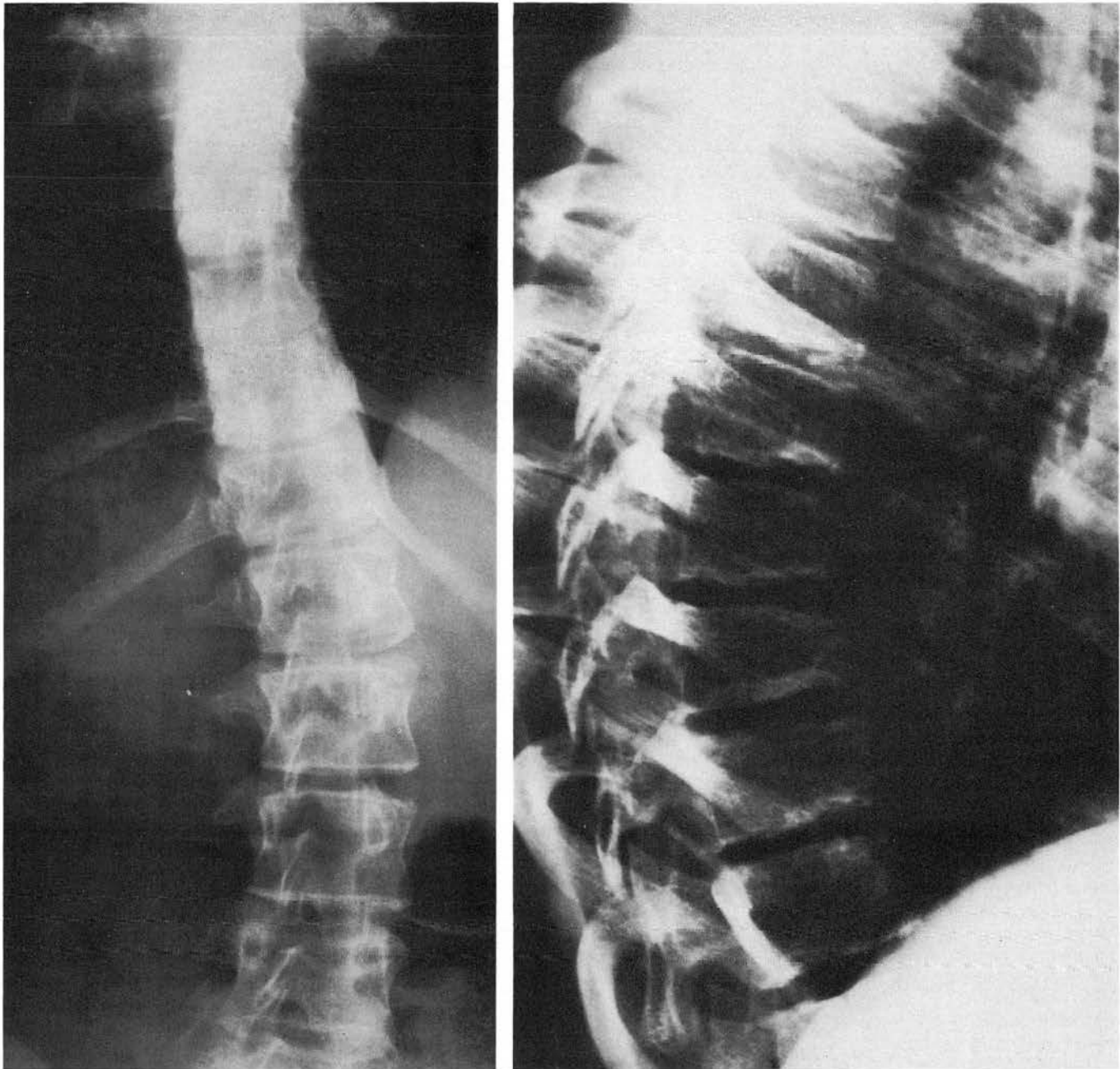


FIG. 4 (a) PA radiograph a patient with Scheuermann's disease showing an "idiopathic-type" scoliosis below the area of kyphosis. (b) Lateral radiograph of the thoracic spine of the same patient showing an increased thoracic kyphosis.

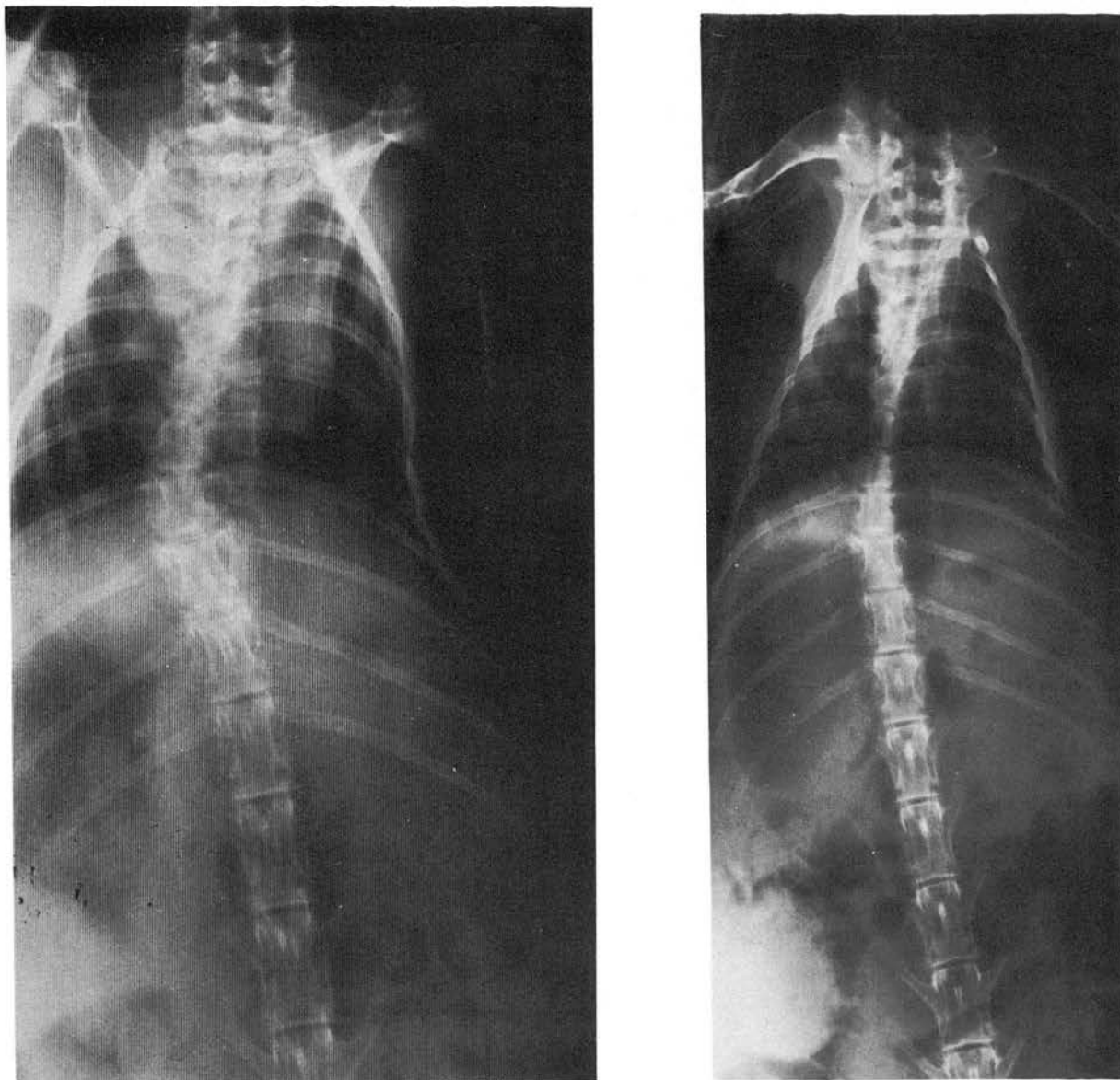


FIG. 5 (a) PA radiograph of a rabbit spine in which experimental idiopathic scoliosis has been produced. (b) PA radiograph of the same spine four weeks after recreation of the thoracic kyphosis. The deformity has considerably improved.

the side (Fig. 4). Interestingly, there was a significant preponderance of females and right sided curves only in the lordoscoliotic group ($P < 0.05$).

EXPERIMENTAL SCOLIOSIS

The crucial nature of the thoracic lordosis was tested in the experimental animal using the growing New Zealand white rabbit as the model. In freshly weaned animals a short segment thoracic lordosis was created by approximating the posterior elements of the lower four thoracic vertebrae using strong suture material. Despite the persistence of the lordosis with growth, a rotational lordoscoliosis was not produced. Rabbits, unlike so many normal children, do not possess any inherent spinal asymmetry in another plane to impart directional instability to the mechanically unstable lordosis. Accordingly the thoracic lordosis produced was then rendered asymmetric by creating at the same time a few degrees of coronal plane asymmetry when the suture material was tightened. In a controlled experiment this biplanar asymmetry was created in ten animals and the subsequent fate of the spine compared with two further groups of animals, one group having coronal plane asymmetry only and the

other the lordosis only. Only in the biplanar group did a progressive lordoscoliosis occur with subsequent growth (Fig. 5).

After the biplanar procedure rupture of the lordosis-maintaining suture occurred in one animal, which spontaneously reconstituted a normal thoracic kyphosis. The early lordoscoliotic deformity resolved over the next four weeks. To confirm this important observation ten animals with biplanar spinal asymmetry had the lordosis-maintaining suture deliberately removed when the lordoscoliotic deformity so produced had reached a magnitude of 30° . Subsequent regression of the deformity occurred in six animals, while in the remaining four the rate of progression was attenuated. Curve magnitude at the time of lordosis release was on average 20° less in those subsequently regressed.

CONCEPT OF A MORE PHYSIOLOGICAL APPROACH TO TREATMENT

From the cadaveric, clinical and experimental studies evolved the concept of restoration of the thoracic kyphosis as an integral part of a surgical procedure aiming to correct all three planes of the idiopathic de-

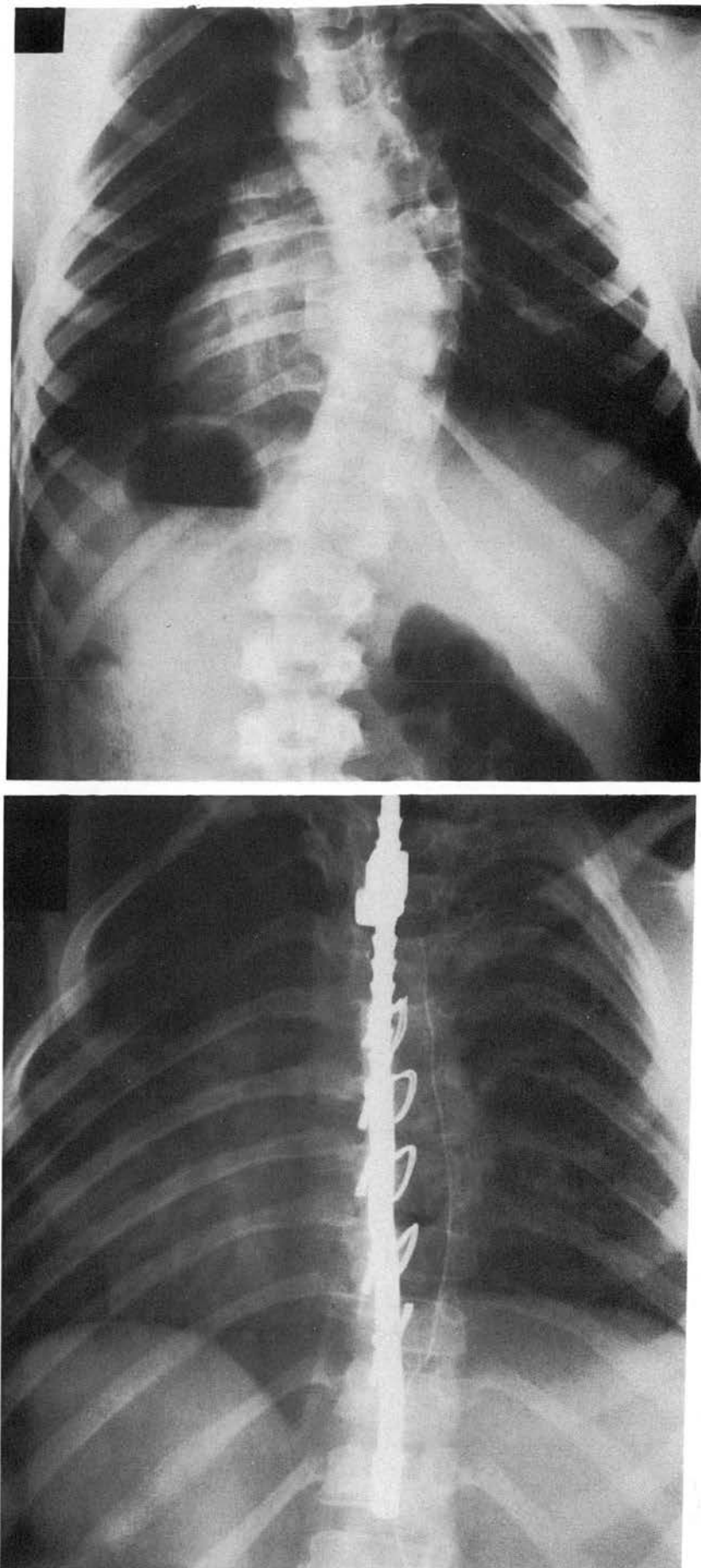


FIG. 6 (a) PA radiograph of a patient with idiopathic thoracic scoliosis before surgery.
(b) PA radiograph of the same patient after concave kyphotic rodding and concave sublaminar wiring. The deformity has been markedly improved.

TABLE 1 Results of 'physiological' treatment

No. of patients	Mean curve size (Degrees)		Correction %	Mean apical rotation (Degrees)		Correction %
	Before	After		Before	After	
20 (Curves < 70°)	46	16	65	28	13	54
5 (Curves > 70°)	100	40	60	46	32	30
25 Total	62	22	65	33	18	45

formity and to possibly allow in the younger child with much growth to go an opportunity for further regression of the secondary deformities of lateral curvature and rotation with the passage of time. The development of segmental spinal instrumentation (11), wiring all the vertebrae in the curve to longitudinal rods, has focussed on the coronal plane, but by bending a longitudinal rod into a normal thoracic kyphotic configuration and drawing the rotated vertebrae to this by tightening concave sublaminar wires enables all three planes of the deformity to be dealt with at one time (12). Necessarily this can only be performed in one stage if the deformity is flexible and not too severe; with significant rigidity a preliminary shortening procedure anteriorly in the nature of multiple disc removal is required to provide room in which the correction can take place and to minimise spinal cord tension. Thus far 25 patients have been operated upon using this technique, 20 with late onset idiopathic scoliosis in whom a posterior spinal fusion has also been performed, and five infantile idiopathic progressive scoliosis in whom posterior fusion has been withheld lest the already too short back of the spine is further tethered in bone. No operative or postoperative complications have been observed in this small group of patients and Table I shows the mean corrections obtained in all three planes. The maximum follow-up period is only 15 months and only one of the four infantile cases, the mildest curve, is showing any evidence of curve regression, and that is minimal. The deformities in the other 21 cases have not changed since the early postoperative position (Fig. 6).

Discussion

This cadaveric, clinical and experimental study emphasises the three dimensional nature of the idiopathic scoliotic deformity and implicates the thoracic lordosis as the essential lesion in idiopathic thoracic scoliosis. The simple mechanical concept of the fundamental nature of the lordosis could not be new and indeed is not, being described 120 years ago by Adams (13), whose classic essay, before x-rays were available, introduced the hypothesis. The precocious minds of Somerville (2) and Roaf (14) lent further support to the lordosis necessitatis; neither could conceive of the deformity occurring in any other way. Indeed Somerville created the first animal model of 'idiopathic' scoliosis in which the lordosis produced must have been rendered asymmetric. Roaf went further and stated that, in terms of simple geometry, if the spinous processes are rotated less than the bodies then there must be a lordosis. He further strictureed, "If kyphosis means an increase in the length of the posterior elements of the vertebral column relative to the anterior elements, the use of the term is certainly erroneous in idiopathic scoliosis." He also made a plea for the study of the morphology of scoliotic spines. In effect, the results of these studies do little more than confirm the views of Adams, Somerville and Roaf, although it is now possible

to explain the clinical behaviour of the idiopathic deformity and its response to treatment (15).

The normal cervical and lumbar lordoses are protected from rotation by considerable available inter-segmental flexion before the limits are reached, by exceedingly strong posterior supporting musculofascial systems, and by the prismatic shape of the vertebral bodies in the transverse plane, whose anteriorly directed bases confer considerable rotational stability (16). By contrast the thoracic vertebral bodies in the transverse plane are prismatic with their apices anteriorly and this potentially unstable situation is protected by a kyphosis with an axis of rotation well in front. Accordingly, in the presence of a thoracic lordosis rotation is inevitable. Non-structural deformities are due to causes extrinsic to the spine, but while a leg length inequality will induce a mild non-progressive lumbar scoliosis by way of compensation, the secondary coronal plane component is applied to an area where there is naturally a lordosis and thus a true lordoscoliosis is produced. This is why children with leg length inequality are detected in such numbers in school screening programmes, which use a forward bending test in order to demonstrate rotation (1).

It is not therefore surprising to find that conservative treatment does not alter the course of the condition of idiopathic scoliosis (5). What would be required to correct the essential lordotic lesion would be an orthosis which would flex the lordosis but this is when the lordosis is rotationally unstable and immediately produces the secondary features of scoliosis and rotation. The kyphotic deformity of Scheuermann's disease being uniplanar and rotationally stable is eminently suitable for conservative treatment and any means which provides thoracic hyperextension can lead to a true physiological improvement of the kyphosis (17). The lordotic deformity of idiopathic thoracic scoliosis can only therefore be effectively treated surgically. The traditional longitudinal distraction techniques of Harrington, with forces applied to the top and bottom of the curve, will only affect the coronal plane component of the deformity and cannot be expected to have much influence on the apical rotation (5). Furthermore, as Roaf also warned, the younger the child when posterior fusion surgery is performed, the more likely the unstable lordosis is to be augmented, thus producing the distinct possibility of further rotational deterioration in the future with growth (14).

The essential nature of the lordosis is confirmed by the animal experiments which show that an asymmetric lordosis readily produces the 'idiopathic-type' deformity with growth. Importantly, when the thoracic kyphosis is reconstituted there is evidence of curve resolution, provided the deformity is not too great and there is plenty of growth to go. The concept of physiological treatment therefore arises, the principle of which is to recreate the thoracic kyphosis which re-sites the axis of spinal rotation in its normally protective position (12). This would clearly have more important implications for the younger child with more growth remaining, in whom instrumental restoration of the kyphosis is performed but fusion is withheld so that subsequent growth will now be more in the patient's favour. There is clearly no need to withhold fusion in the older child with much less growth to go whose newly reformed kyphosis ought to be stabilised in bone.

It was in some ways tempting to leave out the small series of 25 patients with their short follow-up and indeed the main thrust of these investigations concerns more the nature and behaviour of the idiopathic spinal deformity. There are, however, important and serious short term implications derived from these cases. The instrumental advances of Luque in bringing segmental spinal instru-

mentation into the armamentarium of the scoliosis surgeon is primarily referable to the patient with the neuromuscular spinal deformity in whom the greater spinal stability afforded by rods and wires obviates the need for postoperative support (11). Rightly, however, the use of two rods and two sets of sublaminar wires is considered too much of a risk to the spinal cord for the idiopathic deformity in the otherwise normal child. The risks are clearly going to be less if only one rod and one set of sublaminar wires are used and as there is no means via a posterior approach to achieve the necessary correction of the deformity in all three planes the absence of neurological problems or any other complications in this series is important. Furthermore, that there is a safe operative technique that can improve both rotation and lateral curvature by two-thirds as well as restoring the thoracic kyphosis is also important. While the future of the young child with the instrumented spine without fusion is uncertain, the longer the definitive fusion can be postponed, the better will be the final result. For the later onset case, however, the condition is primarily one of deformity and this procedure affords a very satisfactory correction of the rotational prominence with which every patient presents.

References

- 1 Dickson RA. Scoliosis in the community. *Brit Med J* 1983;286:615-8.
- 2 Somerville EW. Rotational lordosis: the development of the single curve. *J Bone Joint Surg (Br)* 1952;34:421-7.
- 3 Blount WP, Schmidt AC. The Milwaukee brace in the treatment of scoliosis. *J Bone Joint Surg (Am)* 1957;39:693.
- 4 Kaiser RP, Shuflebarger HL. The Milwaukee brace in idiopathic scoliosis. Evaluation of 123 completed cases. *Clin Orthop* 1976;118:19-24.
- 5 Aaro S, Dahlborn M. The effect of Harrington instrumentation on the longitudinal axis of rotation of the apical vertebra and on the spinal and rib-cage deformity in idiopathic scoliosis studied by computer tomography. *Spine* 1982;7:457-62.
- 6 Hibbs RA, Risser JC, Ferguson AB. Scoliosis treated by the fusion operation - an end result study of 360 cases. *J Bone Joint Surg (Br)* 1931;13:91-104.
- 7 Lefebvre J, Triboulet-Chassevant A, Missirlu MF. Electromyographic data in idiopathic scoliosis. *Arch Phys Med Rehab* 1961;42:710-11.
- 8 Alexander MA, Season EH. Idiopathic scoliosis: an electromyographic study. *Arch Phys Med Rehab* 1978;59:314-5.
- 9 Deacon P, Flood BM, Dickson RA. Idiopathic scoliosis in three dimensions: a radiographic and morphometric analysis. *J Bone Joint Surg (Br)* 1984;66:8-15.
- 10 Perdriolle R. La scoliose. Son étude tridimensionnelle. Paris: Maloine, 1979.
- 11 Luque ER. Paralytic scoliosis in growing children. *Clin Orthop* 1982;163:202-9.
- 12 Dickson RA, Lawton JO, Archer IA, Butt WP. The pathogenesis of idiopathic scoliosis. *J Bone Joint Surg (Br)* 1984;66:8-15.
- 13 Adams W. Lectures on the pathology and treatment of lateral and other forms of curvature of the spine. London: Churchill & Sons, 1865.
- 14 Roaf R. The basic anatomy of scoliosis. *J Bone Joint Surg (Br)* 1966;48:786-92.
- 15 Dickson RA, Lawton JO, Butt WP. The pathogenesis of idiopathic scoliosis. In: Dickson RA and Bradford DS, eds. Management of spinal deformities. London: Butterworths; 1984.
- 16 Deacon P, Archer IA, Dickson RA. The anatomy and biomechanics of spinal deformity. *J Bone Joint Surg (Br)* 1984;66:289.
- 17 Bradford DS, Moe JH, Montalvo FJ, et al. Scheuermann's kyphosis and roundback deformity. Results of Milwaukee brace treatment. *J Bone Joint Surg (Am)* 1974;56:740-58.

SURGICAL TREATMENT OF LATE-ONSET IDIOPATHIC THORACIC SCOLIOSIS -
THE LEEDS PROCEDURE.

Dickson RA, Archer IA.

Journal of Bone and Joint Surgery; 69B: 709-714, 1987.

The first 50 patients with a minimum follow-up of two years who underwent the Leeds segmental derotation procedure combined with thoracic kyphosis recreation was reported. A 50% correction in the amount of spinal rotation was demonstrated. This was the first technique to reliably untwist the deformed spine and 50% derotation still remains the best reported.

SURGICAL TREATMENT OF LATE-ONSET IDIOPATHIC THORACIC SCOLIOSIS

THE LEEDS PROCEDURE

R. A. DICKSON, I. A. ARCHER

From St James's University Hospital, Leeds

Late-onset idiopathic scoliosis is associated with a rib hump in the thoracic region, and surgery is indicated when this deformity becomes unacceptable. Fifty patients with this deformity were treated by the Leeds procedure, which consists of segmental wiring to a kyphotically-contoured square-ended Harrington rod; this procedure not only derotates the spine but restores the natural thoracic kyphosis, thus avoiding subsequent buckling. All patients were followed up for a minimum of two years. Forty-two of these, who had a pre-operative Cobb angle of less than 60°, were treated by one-stage instrumentation and fusion, while the remaining eight with greater curves underwent preliminary anterior multiple discectomy to provide flexibility with shortening. Postoperative loss of correction was not observed and there were no neurological complications.

There are two varieties of idiopathic scoliosis: early and late onset (Ponseti and Friedman 1950; Dickson 1985). The uncommon, progressive early-onset type is associated with severe deformity and with cardiopulmonary morbidity (Nachemson 1968), while the more prevalent late-onset form is the source of deformity only, there being no organic consequences to the patient (Davies and Reid 1971; Branthwaite, personal communication 1985); the deformity is, however, important, and the greater it is, the more the patient suffers social and psychological deprivation (Bengtsson et al. 1974).

While the early-onset progressive case may be treated conservatively (Mehta and Morel 1979), late-onset idiopathic scoliosis can only be treated effectively by surgery (Miller, Nachemson and Schultz 1984; Dickson 1985). Patients present with asymmetry of the torso, usually in the form of an unacceptable rotational prominence. It is this which the patient wishes to have corrected, and it is remarkable how unsuccessful standard surgical treatment is in achieving this correction. Harrington instrumentation is no more corrective than previous casting methods (Moe and Valuska 1966), and for thoracic curves the average correction is 50% in

the coronal plane and little or nothing in the transverse plane, the rib hump remaining unchanged (Schultz and Hirsch 1973; Aaro and Dahlborn 1982). Posterior segmental wiring systems (Resina and Alves 1977; Luque and Cardoso 1977) are no better (Leatherman et al. 1984) and do not provide effective derotation (Wenger and Carollo 1984). This, together with an unacceptably high complication rate (King 1984), has led to the widely held view that there is no place for two rods and sets of wires in idiopathic scoliosis, and that for patients with this deformity a single Harrington rod and one set of segmental wires is preferable (Leatherman et al. 1984).

That structural scoliosis is as resistant to correction by instrumentation as it was to earlier methods is entirely attributable to its three-dimensional nature. The essential lesion is a lordosis which buckles to the side (Adams 1865; Somerville 1952; Roaf 1966; Dickson et al. 1984). Segmental wiring attached to straight rods, or to rods favouring the coronal-plane component of the deformity, exert their effect behind the axis of the lordosis; such wiring cannot be expected to improve the rotational prominence and may in fact make it worse. Moreover, the tethering effect of posterior surgery in the presence of a lordosis (Roaf 1966) is such that the deformity continues to progress, sometimes at a faster rate than before (Hefti and McMaster 1983).

If the deformity is to be effectively corrected, the pull on the vertebrae in the curve should be backwards rather than to one side; in the thoracic region the

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kyphosis is thereby restored so that the spine is once again in its correct position in relation to the axis of spinal rotation. Subsequent rotational progression is mitigated (Dickson, Lawton and Butt 1984). Sagittal-profile segmental wiring to a Harrington distraction rod is thus the basis of the Leeds technique, which was first performed in November 1982. We report here the results of the first 50 cases of late-onset idiopathic scoliosis treated by this procedure.

PATIENTS AND METHODS

There were 40 females and 10 males with a mean age of 12.5 years (range 9 to 22). Before operation, 42 had a Cobb angle of less than 60° and were treated by a one-stage procedure; the remaining eight with a Cobb angle of between 60° and 90° had a two-stage operation. The deformity was assessed radiographically by measuring the Cobb angle and the angle of apical vertebral rotation (Perdriolle 1979) from erect postero-anterior radiographs obtained just before operation, two weeks thereafter and then at follow-up (average 2 years 3 months; range 2 years to 3 years 3 months).

Operation. The basis of the technique is a standard midline posterior approach to the spine from at least the neutral vertebra above the deformity to the neutral vertebra below; this is followed by the passage of concave sublaminar wires and the insertion of a Harrington distraction rod.

Certain parts of this procedure are critical. For thoracic curves the rod must be bent into at least 20° of kyphosis, held in that position by a square-ended hook-rod relationship. For a particularly rigid curve, the rod may also require bending in the coronal plane (manual pressure medially and downwards over the rib hump determines whether this is necessary). When the rod is inserted it is not distracted until the segmental wires have been tightened. If the rod is distracted first, as in the standard Harrington method, then the whole three-dimensional deformity will become rigid in tension: then either the segmental wires will not achieve their desired aim or the contents of the spinal canal will be put under undue tension. If the rod is still slack at the end of wire-tightening, then and only then is it distracted maximally.

The order of tightening the wires also is critical. The upper and lower ends of the rod are first restrained by local wire-tightening; this also secures the hooks. A rod extending above and below neutral vertebrae is thus preferable. Next, the wires under the apical two vertebrae are tightened, aided by manual pressure downwards and not sideways on to the rotational prominence. This is extremely important in order to achieve maximal derotation, as it is only the apical two vertebrae which are maximally rotated (Dickson et al. 1984). If the wires were tightened above and below the apex before the apical wires, then a significant amount of

the rotational deformity would be "left behind".

The remainder of the wires are then tightened and a posterolateral fusion is performed by interdigitating flaps of bone lifted from the posterolateral elements out to the tips of the transverse processes. Upon this bed, matchsticks of allograft cancellous bone are applied. Unless there is a shortage of bank bone, it is not necessary to use bone from the patient's iliac crest, which necessitates a much longer incision and greater postoperative discomfort. For patients with thoracic curves whose rotational prominence still appears obvious, the apical six ribs on the convex side are divided just distal to the transverse process and the lateral end is tucked anterior to the medial; the rib hump is thereby markedly flattened. A postoperative EDF (elongation-derotation-flexion), polyurethane cast with a zip fastener (Neofract; Johnson & Johnson) is applied 10 days after operation and retained for six months.

The above regimen applies to the typical flexible adolescent idiopathic curve, that is, to those patients with a pre-operative Cobb angle of less than 60° (Figs 1 to 4). For those with bigger, and thus more rigid, deformities (curves between 60° and 90°), a satisfactory correction cannot be achieved by a one-stage posterior procedure, and there is, moreover, a real risk of neurological damage due to tension if such an attempt is made. The deformity needs to be shortened as well as straightened; this is achieved by a preliminary anterior removal of the apical five or six discs and end-plates, thus making considerable space available for the deformity to be "pulled through". Enhanced correction without undue tension is thus obtainable. Two to three weeks later, the procedure of posterior instrumentation described above is then performed (Figs 5 to 8). For curves greater than 90°, and for those previously treated by posterior fusion, we favour a two-stage wedge resection of the spine (Leatherman and Dickson 1979).

RESULTS

The results are shown in Table I, from which it can be seen that, overall, a 65.5% correction of the Cobb angle was achieved; more importantly, however, a 50% correction in rotation was also achieved.

Measurements from radiographs taken two weeks after operation were compared with those at follow-up and indicated that the very small losses of correction had occurred within the first few weeks. There were no neurological complications, no deaths and no infections in this series. Two rod breakages were observed. One occurred in a 15-year-old girl with a 50° curve who, in order to exercise on a trampoline, removed her EDF cast three weeks after operation; the metal implants were taken out and a simple Harrington distraction rod inserted without wires and there have been no further problems. The second rod breakage, in a girl of similar



Fig. 1



Fig. 2



Fig. 3



Fig. 4

A postero-anterior radiograph of a 12-year-old girl (Fig. 1); there is a right thoracic idiopathic scoliosis with 30° of apical rotation, and a right rib hump is clearly seen when she bends forward (Fig. 2). Two years after operation, the apical rotation now measures 10° (Fig. 3), and forward bending shows that good correction of her rib hump has been maintained (Fig. 4).



Fig. 5

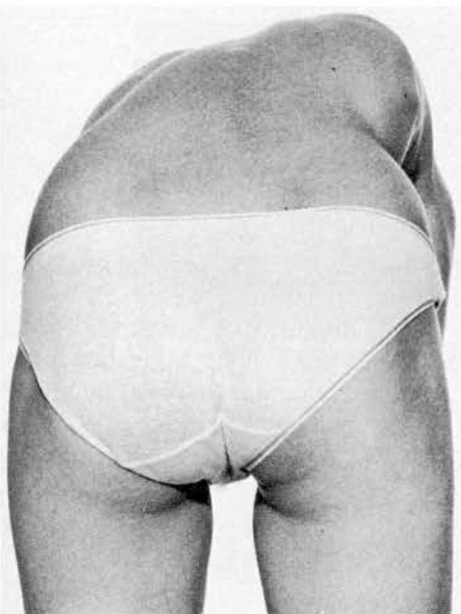


Fig. 6

A postero-anterior radiograph of a 13-year-old girl who has a right thoracic curve with 40° of apical rotation (Fig. 5). Before treatment by a two-stage procedure, forward bending showed a significant rib hump (Fig. 6). Two years later (Fig. 7) apical rotation measured only 20°, and forward bending revealed good correction of the rotational deformity (Fig. 8).

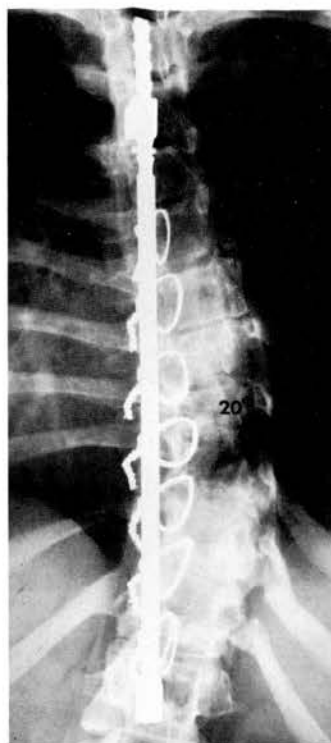


Fig. 7

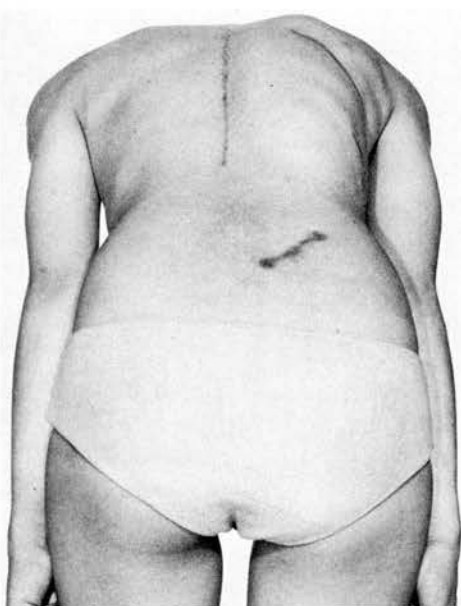


Fig. 8

Table I. Results in 50 patients with late-onset idiopathic scoliosis treated by the Leeds procedure

	Cobb angle <60° (42 patients)			Cobb angle 60°–90° (8 patients)*		
	Before operation	At follow-up	Correction (%)	Before operation	At follow-up	Correction (%)
Mean Cobb angle	54°	18°	66	77°	27°	65
Mean apical rotation	31°	15°	51	54°	27°	50

* Two-stage procedure (see text)

age with a similar curve, was seen in the radiograph taken at two years after the operation; it was not present in the film taken at one year and the patient's initial good correction was sustained and still is. Both breaks occurred at the ratchet–solid rod junction and, because this was associated with a large number of ratchets below the upper hook (an unfavourable mechanical situation), we would recommend keeping the number of ratchets between hooks to a minimum.

DISCUSSION

The indications for operation in late-onset idiopathic scoliosis have become clearer over the past 10 years. Reports that untreated individuals with idiopathic scoliosis have a significant morbidity and mortality from cardiopulmonary compromise (Nachemson 1968; Nilsson and Lundgren 1968; Collis and Ponseti 1969) have been misinterpreted, suggesting that when curves exceed 60° operative treatment is required to prevent chest complications. However, this applies only to early-onset cases (Davies and Reid 1971; Branthwaite, personal communication 1985) and these are now rare. Far more common is late-onset idiopathic scoliosis and, in this variety, once the deformity has become unacceptable, the objective must be to make it acceptable and to keep it so until the risk of subsequent progression has passed. There are thus two important aspects to be considered: correction and maintenance of the correction.

Before the era of spinal instrumentation pioneers in scoliosis surgery obtained their corrections by the use of casts (Risser et al. 1953). While the addition of Harrington instrumentation reduced the rate of pseudarthrosis, there was no improvement in correction (Moe and Valuska 1966), although very few tension neurological complications were observed in the idiopathic case (MacEwen, Bunnell and Sriram 1975). More powerful posterior segmental instrumentation techniques not only failed to produce improved corrections (Kahn 1984; Leatherman et al. 1984) but were associated initially with a neurological complication rate as high as 17% (Luque 1984); even with further not inconsiderable experience, this rate had dropped to only 4% (King 1984).

The failure to achieve correction of rotation and the perilously high rate of neurological complications are both readily explained by the three-dimensional nature of structural scoliosis. Before the advent of radiographs,

the buckled lordosis of the deformity was quite clear to those who studied it (Dick 1864; Adams 1865). The taking of radiographs, however, while enabling the bony spinal column to be visualised, artificially highlighted the lateral spinal curvature; despite the occasional warning (Somerville 1952; Roaf 1966), this aspect of the deformity thereafter attracted all the attention and therapeutic effort.

The futility of longitudinal distraction in correcting rotation has long been known (Schultz and Hirsch 1973) and it would thus appear superficially tempting to pull each vertebra horizontally to the midline by segmental wiring in an effort to improve correction. However, the lordotic apex of the deformity lies in front of the axis of spinal rotation and therefore not only can the rotational deformity not be improved but may in fact be made worse. Only by segmentally pulling the deformity posteriorly in relation to the axis of spinal column rotation can derotation be achieved and, for thoracic curves, the restoration of kyphosis is an integral part of the derotation process. This has nothing to do with the preservation of physiological sagittal curves, often adduced as the reason for slightly bending the longitudinal rods (Luque 1984), but is the essential manoeuvre in producing true derotation (Dickson et al. 1984).

Understanding the nature of the apical rigidity helps to clarify the necessary process of correction and what must be done if rigidity is excessive. The thoracic facet joints are obliquely disposed and favour segmental rotation about an axis in the vertebral body anteriorly. In the presence of a thoracic lordosis, however, the axis of rotation is situated posteriorly, at about the level of the facet joints whose oblique disposition resists segmental rotation to produce a local rigid area. As the apical region is drawn backwards, the axis of rotation comes to lie more and more in front of the facets with rigidity becoming progressively less. Thus recreation of the thoracic kyphosis positively enhances correctability.

As curve size increases, so the ability of posterior surgery alone to correct the curve diminishes, and a more aggressive approach becomes necessary. Our clinical experience suggests that a figure of 60° is appropriate as a threshold above which an anterior space-making procedure is necessary; for a curve of up to 90° this can be performed at disc level. Neurological problems are produced, not by the sublaminar site of the wires, but by the fact that they may actually increase the deformity if

no anterior first-stage procedure is performed.

It can thus be seen that the Leeds procedure is really applicable to curves not in excess of 90°. The 50% correction of the rotational component obtained in our series is most rewarding, and such a figure has not hitherto been achieved. The ingenious method of Dubousset and his colleagues achieves a correction of rotation of 40%; their patients all had mild flexible curves (Dubousset et al. 1986). While this procedure also specifically addresses the sagittal plane, the spine is only derotated where the hooks are sited and thus the apical region is "left behind". As the apical region is the area of greatest rotation, ideally it should be tackled by the instrumentation and, indeed, should be tackled first.

With late-onset idiopathic scoliosis the risk of subsequent rotational progression is less than with the early-onset type, there being less time for the spine to deform before maturity. A posterolateral spinal fusion is therefore performed at the time of posterior instrumentation. The spine does not stop growing until, on average, 10 years after the rest of the skeleton (Haas 1939; Bernick and Cailliet 1982), and a fused thoracic kyphosis is a much safer sagittal profile than a persistently tethered lordosis. With rod and wires in the spine, the available surface area for producing fusion is correspondingly reduced. The addition of bone graft material is thus advisable, although it does not matter whether this is allograft or autograft (Aurori et al. 1985).

It is the three-dimensional nature of the deformity of idiopathic scoliosis which holds the key to successful correction. In particular it is the rigid apical lordosis which has to be unravelled: if deformity is to be rendered acceptable without the risk of neurological damage, then bigger curves do need more aggressive surgery. With such curves a planned and staged treatment programme is safer than a one-stage posterior operation.

REFERENCES

- Aaro S, Dahlborn M. The effects of Harrington instrumentation on the longitudinal axis rotation of the apical vertebra and on the spinal and rib-cage deformity in idiopathic scoliosis studied by computer tomography. *Spine* 1982;7:456-62.
- Adams W. *Lectures on the pathology and treatment of lateral and other forms of curvature of the spine*. London: J Churchill & Sons, 1865.
- Aurori BF, Weierman RJ, Lowell HA, Nadel CI, Parsons JR. Pseudarthrosis after spinal fusion for scoliosis: a comparison of autogeneic and allogeneic bone grafts. *Clin Orthop* 1985; 199:153-8.
- Bengtsson G, Fällström K, Jansson B, Nachemson A. A psychological and psychiatric investigation of the adjustment of female scoliosis patients. *Acta Psychiatr Scand* 1974;50:50-9.
- Bernick S, Cailliet R. Vertebral end-plate changes with ageing of human vertebrae. *Spine* 1982;7:97-102.
- Collis DK, Ponseti IV. Long-term follow-up of patients with idiopathic scoliosis not treated surgically. *J Bone Joint Surg [Am]* 1969;51-A:425-45.
- Davies G, Reid L. Effect of scoliosis on growth of alveoli and pulmonary arteries and on right ventricle. *Arch Dis Child* 1971;46:623-32.
- Dick H. A new instrument for the treatment of spinal curvature. *Medical Times and Gazette* 1864;11:203-4.
- Dickson RA, Lawton JO, Butt WP. The pathogenesis of idiopathic scoliosis. In: Dickson RA, Bradford DS, *Management of spinal deformities*. Orthopaedics 2. London, etc: Butterworths, 1984:1-37.
- Dickson RA, Lawton JO, Archer IA, Butt WP. The pathogenesis of idiopathic scoliosis: biplanar spinal asymmetry. *J Bone Joint Surg [Br]* 1984;66-B:8-15.
- Dickson RA. Conservative treatment for idiopathic scoliosis. *J Bone Joint Surg [Br]* 1985;67-B:176-81.
- Dubousset J, Graf H, Miladi L, Cotrel Y. Spinal and thoracic derotation with CD instrumentation. *Orthop Trans* 1986;10:36.
- Haas SL. Growth in length of vertebrae. *Arch Surg* 1939;38:245-9.
- Hefti FL, McMaster MJ. The effect of the adolescent growth spurt on early posterior spinal fusion in infantile and juvenile idiopathic scoliosis. *J Bone Joint Surg [Br]* 1983;65-B:247-54.
- Kahn A III. Segmental spinal instrumentation in idiopathic scoliosis. In: Luque ER, ed. *Segmental spinal instrumentation*. Thorofare, NJ: Slack Inc, 1984:147-63.
- King AG. Complications in segmental spinal instrumentation. In: Luque ER, ed. *Segmental spinal instrumentation*. Thorofare, NJ: Slack Inc, 1984:301-30.
- Leatherman KD, Dickson RA. Two-stage corrective surgery for congenital deformities of the spine. *J Bone Joint Surg [Br]* 1979;61-B:324-8.
- Leatherman KD, Johnson JR, Holt RT, Roadstone P. A clinical assessment of 357 cases of segmental spinal instrumentation. In: Luque ER, ed. *Segmental spinal instrumentation*. Thorofare, NJ: Slack Inc, 1984:165-84.
- Luque ER, Cardoso A. Segmental correction of scoliosis with rigid internal fixation: preliminary report. *Orthop Trans* 1977;1:136-7.
- Luque ER. State of the art. In: Luque ER, ed. *Segmental spinal instrumentation*. Thorofare, NJ: Slack Inc, 1984:1-11.
- MacEwen GD, Bunnell WP, Sriram M. Acute neurological complications in the treatment of scoliosis: a report of the Scoliosis Research Society. *J Bone Joint Surg [Am]* 1975;57-A:404-8.
- Mehta MH, Morel G. The non-operative treatment of infantile idiopathic scoliosis. In: Zorab PA, Siegler D, eds. *Scoliosis 1979*. Based on the proceedings of the sixth symposium on scoliosis held at the Cardiothoracic Institute, Brompton Hospital, London, on September 17 and 18, 1979. London: Academic Press, 1980:71-84.
- Miller JA, Nachemson AL, Schultz AB. Effectiveness of braces in mild idiopathic scoliosis. *Spine* 1984;9:632-5.
- Moe JH, Valuska JW. Evaluation of treatment of scoliosis by Harrington instrumentation. *J Bone Joint Surg [Am]* 1966; 48-A:1656-7.
- Nachemson A. A long term follow-up study of non-treated scoliosis. *Acta Orthop Scand* 1968;39:466-76.
- Nilsson U, Lundgren K-D. Long-term prognosis in idiopathic scoliosis. *Acta Orthop Scand* 1968;39:456-65.
- Perdriolle R. *La scoliose: son étude tridimensionnelle*. Paris: Maloine, 1979.
- Ponseti IV, Friedman H. Prognosis in idiopathic scoliosis. *J Bone Joint Surg [Am]* 1950;32-A:381-95.
- Resina J, Alves AF. A technique of correction and internal fixation for scoliosis. *J Bone Joint Surg [Br]* 1977;59-B:159-65.
- Risser JC, Lauder CH, Norquist DM, Craig WA. Three types of body casts. *Am Acad Orthop Surg Instruct Course Lect* 1953;10:131-42.
- Roaf R. The basic anatomy of scoliosis. *J Bone Joint Surg [Br]* 1966;48-B:786-92.
- Schultz AB, Hirsch C. Mechanical analysis of Harrington rod correction in idiopathic scoliosis. *J Bone Joint Surg [Am]* 1973;55-A:983-92.
- Somerville EW. Rotational lordosis: the development of the single curve. *J Bone Joint Surg [Br]* 1952;34-B:421-7.
- Wenger D, Carollo J. Biomechanics of segmental spinal instrumentation. In: Luque ER, ed. *Segmental spinal instrumentation*. Thorofare, NJ: Slack Inc, 1984:31-48.

THE KYPHOTIC SPINE IN MYELOMENINGOCELE.

Dickson RA, Leatherman KD.

In 'Myelomeningocele' (McLaurin RL Ed) Grune and Stratton:
New York, 609-620, 1977.

This publication and the next four (31, 32, 33, 34) are presented to show a broad and long standing experience of different types of spinal deformity and their management.

THE KYPHOTIC SPINE IN MYELOMENINGOCELE

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INTRODUCTION

Congenital lumbar kyphosis is the least common spinal deformity encountered in patients with myelomeningocele. It differs from the usual paralytic curvature by being rigid, structural from the outset, and present at birth. This latter characteristic makes closure of the overlying sac extremely difficult. Notoriously resistant to any form of conservative therapy all congenital kyphoses are the subject of surgical intervention. It is tempting to approach these posterior deformities from the back, and indeed, the early results from doing so may appear rewarding. Unfortunately, there is an innate tendency for these lumbar kyphoses to progress despite posterior surgery. This is because the basic pathology exists anteriorly and unless the operative procedure is performed with this in mind, results are likely to continue to be disappointing.

PATHOLOGY

The excellent work of Hoppenfield,² although frequently referred to, is often overlooked. In a meticulous study involving clinical and radiographic investigations correlated with cadaver dissections he clearly demonstrated the true

deforming forces. Figures 1 and 2 show these anterior basic mechanisms. The apical vertebral body is wedged anteriorly and often appears 'rounded off'. The vertebrae above and below share in this structural bony deformity but to a lesser extent. Frequently, as in a high level neurological lesion, there is extensive loss of the posterior elements which complements the anterior changes. Soft tissues are an integral part of the anterior deforming forces, in particular tight annuli fibrosi and a contracted anterior longitudinal ligament. Indeed when Hoppenfield divided these in cadaver specimens he could obtain a good correction. To these must be added three sets of muscles which are also offenders. Due to posterior bony element rotation the paraspinal muscles convex to exert a flexor effect on the already deformed spine. The crura of the diaphragm and the psoas musculature are the others responsible. It is hardly surprising, therefore, that surgical intervention from the back is attended by poor results when the problems exist in front.

CLINICAL FEATURES

Myelomeningocele with kyphosis is always associated with severe lower limb paralysis and in Sharrard's experience the commonest situation is paralysis of all muscles below the third lumbar neural segment. Many, however, have neurological levels of thoracic twelve or above with associated hydrocephalus and, therefore, have no walking potential.

The clinical appearance of the characteristic deformity is shown in Figure 3. The typical short-segment kyphosis confined to the lumbar spine can be clearly seen in this five year old, Figure 3-A. The overlying skin is always scarred and of poor quality, Figure 3-B. In this instance the integument is intact but frequently skin breakdown with

super-added infection lead to ulceration which may resist conservative treatment. Infection may then spread to the cerebro-spinal fluid and threaten the neurological level or even life itself.

While the most severe deformities are accompanied by a compensatory thoracic lordosis, the most usual situation is that the entire spine above angulates further forward as the kyphosis progresses until chest and thighs meet which may be the only factor preventing the child from toppling over.

MANAGEMENT

There is no place for bracing alone in the management of these individuals whose deformities are rigid and progressive. Surgery is the only solution to this problem. It would seem logical to correct the spinal lesion at the same time as the defect is closed on the first day of life and, indeed, Sharrard⁵ has developed a technique of neonatal osteotomy-resection for this purpose. This may be helpful by enabling good skin cover to be achieved from the outset and would lessen the tendency to recurrent breakdown and ulceration which would otherwise occur in the poor quality skin stretched by the kyphotic deformity. All too frequently, however, the child reaches the spinal center long after this latter situation has developed. The surgeon is then driven into operative treatment for two reasons - 1) to prevent or definitively treat the overlying skin problem, and 2) to prevent or correct the underlying deformity and so enable the child to be an independent sitter.

The surgical approaches to these two problems are totally different and it is extremely important that this point be appreciated. While we pay particular attention to skin asepsis in spinal surgery this may be extremely

difficult or even impossible to achieve. Indeed the excision of an overlying septic skin ulcer may be the only route of approach to the underlying deformity. For this reason the amount of exposure and quantity of implant materials inserted must be kept to a minimum. Figure 4 illustrates such a case. A 140 degree lumbar kyphosis was associated with overlying skin ulceration resistant to non-operative local treatment. The spine was approached posteriorly by excising the ulcerated skin and the redundant spinal cord ligated and excised. A wedge excision of the apical vertebral body was then performed and compression hooks were used for fixation (Fig. 4-B). A good correction was obtained and the overlying wound healed uneventfully in an area of now good quality skin. When the child attempted to sit, the spine above collapsed forward, the compression hooks tented the skin and had to be removed. The deformity is recurring, (Fig. 4-C), and the child is still unable to sit. This must be the expectation when only a local fusion is performed after wedge excision with limited fixation from the back when the deforming forces exist in front.

When the deformity is not associated with an overlying skin problem then definitive corrective surgery can be performed, bearing in mind the pathological anatomy in these spines. Figure 5 illustrates this point. This child with a 100 degree lumbar kyphosis was unable to sit without an anterior support to prevent his toppling over (Fig. 5-A). A two stage surgical procedure was performed. In the first stage the kyphosis was approached anteriorly, the tight anterior longitudinal ligament and annuli fibrosi divided, the deformed vertebra removed, and an anterior rib strut graft inserted. At a second operation two weeks later the spine was approached posteriorly, the wedge excision closed

with a compression system, and an extensive posterior fusion was performed with Harrington distraction rods extending from the middle of the thoracic spine to the sacrum. The lateral radiograph (Fig. 5-B) shows the position of the anterior strut graft and a good correction of the deformity achieved. At follow-up four years later (Fig. 5-C) the correction has been maintained, the anterior graft has been incorporated, there is a solid posterior fusion with hooks and rods holding, and the patient can sit upright independently with a straight spine.

DISCUSSION

Congenital lumbar kyphosis in myelomeningocele is characterized by its severity, rigidity, and progression. Superimposed on the structural vertebral changes are strong anterior soft-tissue deforming forces. The sitting potential of the child, already a non-walker, is in serious jeopardy as his center of gravity is drawn further anteriorly. Furthermore, problems concerning the quality of the skin overlying the deformity are an ever-present threat to life, there being a high incidence of cerebro-spinal infection in these individuals. Neonatal osteotomy-resection of the deformed vertebra may solve the immediate problem of providing a good quality to the overlying integument and this must be the initial treatment of choice. Recurrence of deformity and non-union are known complications, however, but this procedure affords the only means of preventing skin sepsis. With healthy skin cover the child can then await the definite corrective surgery he will require when more mature to prevent recurrence of deformity which is a sine qua non of gravity's further adverse influence on the anterior deformity forces.

Unfortunately, many of these children do not have the opportunity for this type of surgery at birth and present much later to a spinal center with established skin ulceration. Local posterior wedge excision of the apex of the deformity should be performed in order to allow satisfactory skin healing.⁶ Such a procedure cannot be expected to prevent progression of deformity and it would, therefore, appear more prudent not to further endanger the situation by introducing a foreign body (wire, screw, staple) which in any event will produce no beneficial effect. The introduction of an implant should await the definite corrective two stage procedure which can be performed when the skin is healthy and the spine more mature. A two stage anterior and posterior approach has already proved itself effective in the treatment of other severe, rigid, spinal deformities^{1,3,4} and is the only method of dealing with the anterior deforming forces. Both anterior and posterior fusions are essential in producing stability in paralytic spinal disorders and in order to prevent the spine from collapsing above and below the corrected kyphosis, a solid posterior fusion is necessary extending well above the deformed area down to the sacrum.

The suggested management of kyphosis in myelomeningocele is summarized in Table 1.

REFERENCES

1. Dickson, R.A. and Leatherman, K.D.: Spinal deformity in adults - changing concepts. *J. Bone Joint Surg.*, 58A In press, 1976.
2. Hoppenfield, S.: Congenital kyphosis in myelomeningocele *J. Bone Joint Surg.*, 49B:276, 1967.

3. Leatherman, K.D.: Resection of vertebral bodies.
J. Bone Joint Surg., 51A:206, 1969.
4. Leatherman, K.D.: The management of rigid spinal curves.
Clin. Orthop, 93:215, 1973.
5. Sharrard, W.J.W.: Spinal osteotomy for congenital
kyphosis in myelomeningocele. *J. Bone Joint Surg.*,
50B:466, 1968.
7. Sharrard, W.J.W. and Drennan, J.C.: Osteotomy-excision
of the spine for lumbar kyphosis in older children with
myelomeningocele. *J. Bone Joint Surg.*, 54B:50, 1972.

TABLE 1.
Congenital Kyphosis in Myelomeningocele

<u>Management</u>	
<u>Neonatal Osteotomy-Excision</u>	- On the first day of life to aid sac closure and provide adequate skin cover
<u>Posterior Wedge Excision</u>	- To provide healthy skin cover when ulceration of the integument overlies the kyphosis
<u>The Two Stage Procedure</u>	- Necessary in all cases to provide definitive correction of the deformity

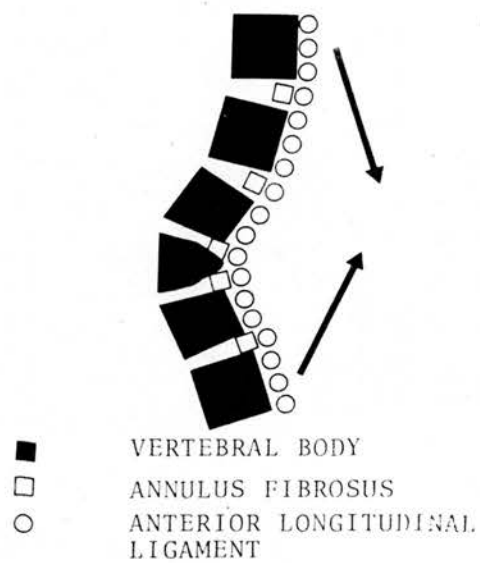


Fig. 1. The anterior deforming forces.



Fig. 2. Lateral radiograph of a congenital lumbar kyphosis.



Fig. 3A. The characteristic clinical deformity.

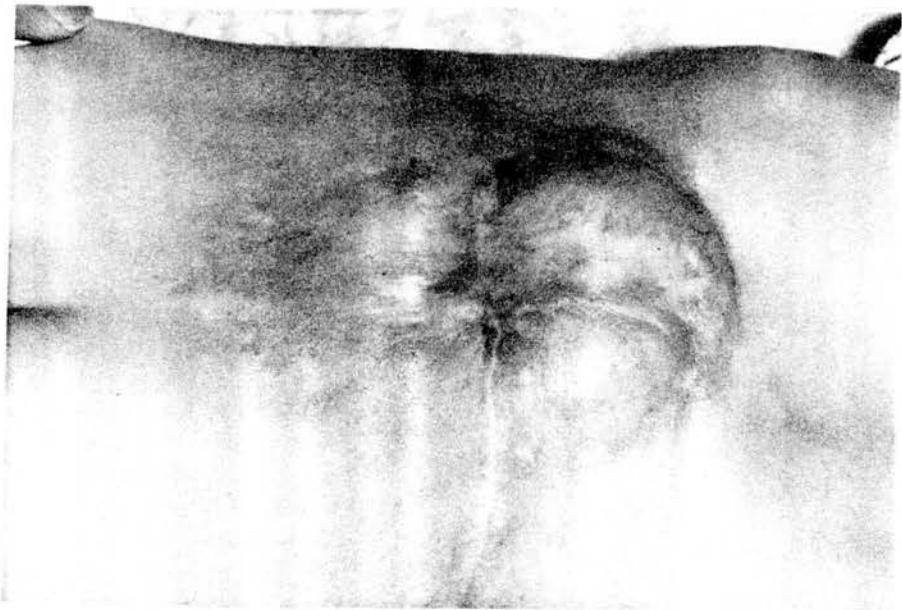


Fig. 3B. The overlying skin is scarred and of poor quality.

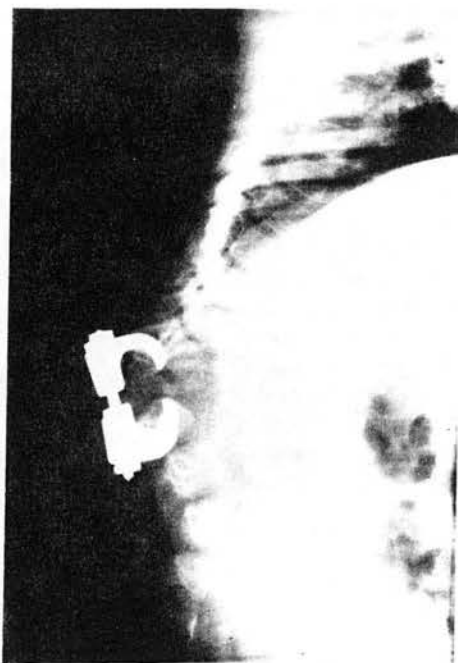


Fig. 4A (top left). Pre-operative lateral radiograph of a severe 140 degree congenital lumbar kyphosis.

Fig. 4B (top right). Lateral radiograph after posterior wedge excision and internal fixation with compression hooks. A good correction has been achieved.

Fig. 4C (bottom left). When the spine collapsed forward above the kyphosis, the hooks had to be removed to prevent skin breakdown. The deformity is recurring and the child cannot sit.

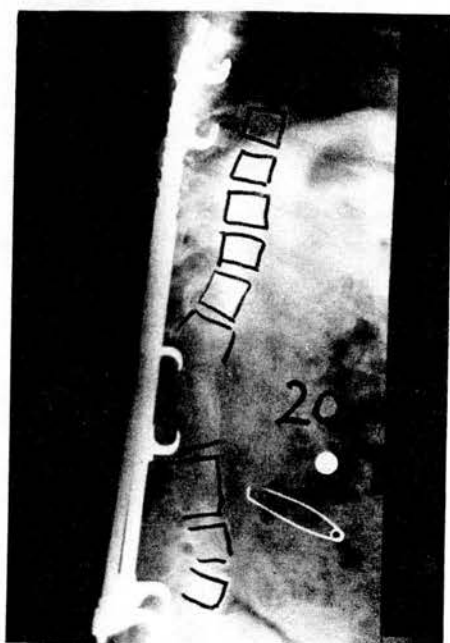
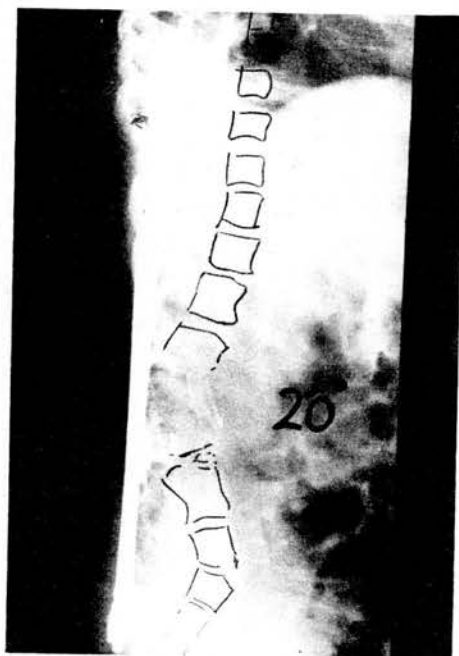
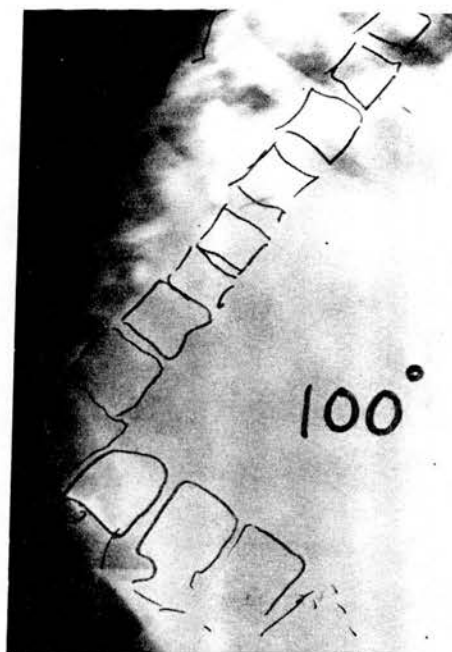


Fig. 5A (top left). Pre-operative lateral radiograph of a severe 100 degree congenital lumbar kyphosis.

Fig. 5B (top right). Lateral radiograph showing the excellent correction achieved by a two-stage procedure—anterior soft tissue release, vertebral body resection, and posterior fusion with rods.

Fig. 5C (bottom left). Lateral radiograph four years later. Hooks and rods are holding and there has been no loss of correction.

VERTEBRAL BODY RESECTION FOR SPINAL DEFORMITY.

Dickson RA, Leatherman KD.

In 'Myelomeningocele' (McLaurin RL Ed) Grune and Stratton:
New York, 621-633, 1977.

VERTEBRAL BODY RESECTION FOR SPINAL DEFORMITY IN MYELOMENINGOCELE

Robert A. Dickson, Ch.M., F.R.C.S.
and Kenton D. Leatherman, M.D.

INTRODUCTION

The management of scoliosis, kyphosis, and lordosis in relation to myelomeningocele has already been well described in previous chapters. However, the axial skeleton in spina bifida may not just be the site of a progressive collapsing type of deformity. It is in the spine of the myelodysplastic that some of the most severe problems exist which are among the most challenging in the entire field of spinal surgery and which warrant the most exacting operative intervention. The two-stage vertebral body resection and posterior fusion with Harrington instrumentation^{3,4} is the only satisfactory method of dealing with the most complex situations.

THE CONCEPT OF VERTEBRAL BODY RESECTION

The removal of a vertebral body is not a new operation. It was pioneered many years ago in Hong Kong by Professor Hodgson for tuberculous spinal disease,² and in Louisville for the management of rigid spinal deformities.^{3,4} Its importance in the management of severe deformities in spina bifida is only now being appreciated in other centres. Fig. 1 shows the frequency of the various different operative procedures used to deal with myelomeningocele spines in our

centre. It can be seen that almost two-thirds of the procedures performed involved excision of a vertebral body. Posterior fusion with rods, by far the most popular operation for idiopathic scoliosis, was used in less than one-third of our patients. But why the apparent concentration on vertebral body resection, a procedure for rigid deformities, when the basic lesion is supposed to be a paralytic collapsing type of curve? In our experience of over 50 cases of spinal deformity in myelomeningocele 47 percent had associated congenital abnormalities, the most common being a unilateral unsegmented bar. A rigid spinal curvature, therefore, frequently complicates the issue, and requires a more aggressive approach than if the deformity was merely paralytic in origin.

PATIENTS

Eleven patients underwent vertebral body resection for deformities excluding congenital lumbar kyphosis. The mean age of these individuals at operation was 13.4 years and the mean follow-up period of 3.5 years. Fig. 2 shows the various deformity types. Six patients had scoliosis as the sole deformity, three had an associated lordosis, and two an associated kyphosis.

MANAGEMENT

Table 1 shows the various operative procedures of which vertebral body resection was an integral part. Vertebral body resection alone was only performed in two cases. Seven patients underwent a second-stage posterior fusion with rods (usually with posterior osteotomy), and one required Dwyer instrumentation in addition. In one individual with a double structural curvature two vertebral body resections were

performed, and the posterior fusion with rods was the finale of a three-stage adventure. The two-stage vertebral body resection followed by posterior fusion with Harrington instrumentation was, therefore, the most usual performed. The value of this procedure developed in Louisville is illustrated by Fig. 3. This five year old has a typical pre-operative radiographic appearance, Fig. 3-A. This is a supine film of a child unable to sit. A lower left thoracic unilateral unsegmented bar has given rise to this severe 132 degree deformity. The left costal margin is tucked under the left iliac wing which has migrated superiorly due to the marked degree of pelvic tilt. At the first stage the apical vertebral body is approached through the bed of the rib above the apex on the convex side, in this instance the right eighth rib. This vertebral body is then removed, Fig. 3-B. At the second stage, usually performed two weeks later, the posterior bony elements, if present, corresponding to the level of the body excised, are then removed and this area is closed with a compression system. An extensive posterior fusion is then performed from the middle of the thoracic spine right down to the sacrum, Fig. 3-C. The deformity, now corrected to 87 degrees is maintained by means of a distraction rod which must extend well above the congenital component of the deformity and right down to the sacrum to get below the paralytic component. The special sacral alar hook is ideal for this purpose and obviates the need for the troublesome sacral bar. The pelvic tilt has been markedly reduced. At follow-up eighteen months later this child, with no leg function, is an independent sitter.

Sometimes it is necessary to be even more aggressive, particularly when two severe rigid curvatures exist in the same spine. Fig. 4 illustrates such a case. This three

year old girl has an upper left thoracic unilateral unsegmented bar giving rise to a severe 110 degree deformity, Fig. 4-A. Below this there is a 112 degree thoraco-lumbar curve which radiographically defies precise classification but whose absence of movement on lateral bending confirms its rigid character. A severe pelvic tilt is again present in this non-sitter. At the first stage, Fig. 4-B, the upper apical vertebral body was removed through the bed of the right fifth rib. Two weeks later at the second stage, Fig. 4-C, the lower apical body was excised subpleurally and retroperitoneally via the eleventh left rib. At the third stage two further weeks later, Fig. 4-D, an extensive posterior spinal fusion with Harrington instrumentation was performed. The site of the upper body resection was closed with a compression system on the convex side. Two distraction rods were used to jack up the concavity of the lower curve, it being technically extremely difficult to fit a compression system into non-existent posterior elements. At follow-up one year later, Fig. 4-E, hooks and rods are holding, there has been no loss of correction, and the child can sit with a clinically straight spine.

It is tempting when excision of a vertebral body has given rise to a good correction to omit the second stage. This temptation must be resisted. Fig. 5 serves as the necessary warning. A severe 100 degree curvature, Fig. 5-A, could only be corrected to 60 degrees by halo-femoral traction, Fig. 5-B. The third lumbar vertebral body was then removed and the patient retained in traction. The deformity was, thereby, further corrected to 37 degrees, Fig. 5-C, and a spontaneous interbody fusion occurred. No second stage was performed but two years later the curvature has increased to 60 degrees, Fig. 5-D.

Another operation was clearly indicated.

RESULTS

Table 2 summarizes the results of vertebral body resection for spinal deformity in myelodysplasia in our centre. The mean pre-operative curvature of these patients was 97 degrees and operative treatment corrected this to 55 degrees (a 43 percent correction). In one individual the wound took four weeks to heal, and one case progressed again after surgery. There were no infections, no cases of increased neurological loss and no deaths in this series. The absence of infection we believe to be due to our policy of prophylactic antibiotics, urinary sterilization, and meticulous pre- and per-operative skin care.

Although operative technique is particularly important in spinal surgery, the operation itself is only a part of the overall management of these children. The resection of a vertebral body may appear to be a formidable procedure but, performed properly, it is not attended by a high complication rate. We have looked very carefully at the post-operative course of patients undergoing spinal surgery for deformities associated with myelodysplasia and have painstakingly documented the important variables. It can be seen from Table 3 that with regard to our two-stage procedure the first stage (vertebral body resection) was associated with a blood loss of 600 cc's, less than half of that associated with posterior fusion with rods. This we believe to be due to removing the vertebral body slice by slice with a chisel and then immediately covering the raw surface with bone wax. By this means bleeding is kept to a minimum from this extremely vascular cancellous bone. Furthermore, if blood loss is replaced as it occurs we have never found it

necessary to infuse additional blood in the post-operative period following vertebral body resection. In contradistinction even when blood loss is replaced during posterior fusion with rods we have found it necessary in one-third of all cases to give an additional unit on the third post-operative day. This is based arbitrarily on not allowing the haemoglobin to fall below 10 G. in the post-operative phase.

It is important to realize that in these children with abnormalities of function involving many systems, post-operative problems are more likely to arise than if the child was otherwise normal. It is the early complications, if not promptly treated, that are life-threatening. Table 4 is taken from our study of all spinal surgery performed on children with spina bifida,¹ not just the ones who have undergone body resection. With the exception of the one case of prolonged cerebro-spinal fluid leak in an individual who underwent excision of a diastematomyelia in addition to spinal stabilization, all these early complications are those associated with surgery in general, not just spinal surgery. Our surgical forefathers have warned us that any of these may be associated with a rise in temperature in the post-operative phase. However, all patients undergoing spinal surgery for myelodysplasia have a post-operative pyrexia with a mean maximum temperature of 101.7 degrees Fahrenheit and mean duration until this falls to normal of eight days. The mean maximum temperature of those who developed early post-operative complications did not differ significantly from those who had uneventful post-operative courses, nor was there any significant trend for the temperature to be elevated above the mean. The time taken for the temperature to revert to normal was not dissimilar

in the two groups. Repeated careful clinical examinations of all post-operative patients are, therefore, mandatory as inspection of the temperature chart is unreliable.

CONCLUSIONS

These are some of the most aggressive forms of surgical intervention that can be performed on the spine. We believe that there is a world of difference between being condemned to a supine life and being an independent wheel-chair operating sitter. The pleasure of retained eyesight is one of the few remaining functions in these individuals but a twenty-four hour a day inspection of the ceiling of their room can hardly be described as visual satisfaction. An axial skeleton which points upwards on sitting allows the quality of life to assume a new dimension in these most unfortunate children.

REFERENCES

1. Dickson, R.A. and Leatherman, K.D.: Spinal Deformity in Myelomeningocele - Surgical Treatment. In press, 1976.
2. Hodgson, A.R.: Correction of fixed spinal curves. *J. Bone Joint Surg.*, 47A:1221, 1965.
3. Leatherman, K.D.: Resection of vertebral bodies. *J. Bone Joint Surg.*, 51A:206, 1969.
4. Leatherman, K.D.: The management of rigid spinal curves. *Clin. Orthop.*, 93:215, 1973.

TABLE 1.

Type of Procedure

Body Resection and Posterior Fusion with Rods	7
Body Resection Only	2
Two Body Resections and Posterior Fusion with Rods	1
Body Resection, Dwyer, and Posterior Fusion with Rods	1

TABLE 2.

Vertebral Body Resection

Mean Preop. Curve	97
Mean Postop. Curve	55
Early Complications - Delayed Wound Healing	1
Late Complications - Pseudarthrosis/Marked Loss of Correction	1
Infections	0
Increased Neurological Loss	0
Deaths	0

TABLE 3.
Blood Loss

<u>Body Resection</u>	<u>Posterior Fusion With Rods</u>
600 cc's	1340 cc's
No Additional Blood Required	Additional Blood Frequently Required on Third Day

TABLE 4.
Early Complications

Delayed Wound Healing	3
Haematoma	1
Vaginal Discharge	1
Urinary Infection	1
Atelectasis	1
Pneumothorax	1
C S F Leak	1

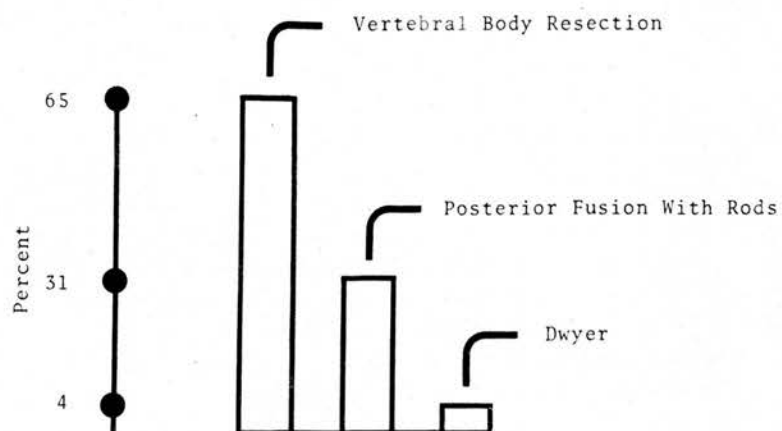


Fig. 1. The frequency of the various operative procedures.

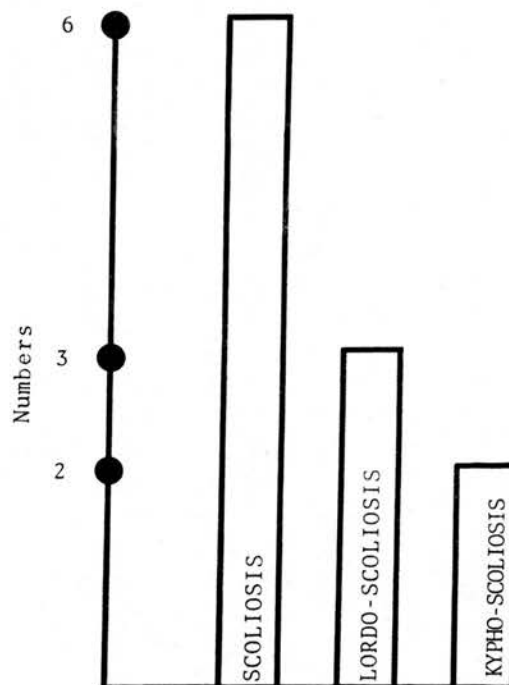


Fig. 2. The frequency of the various deformity types.

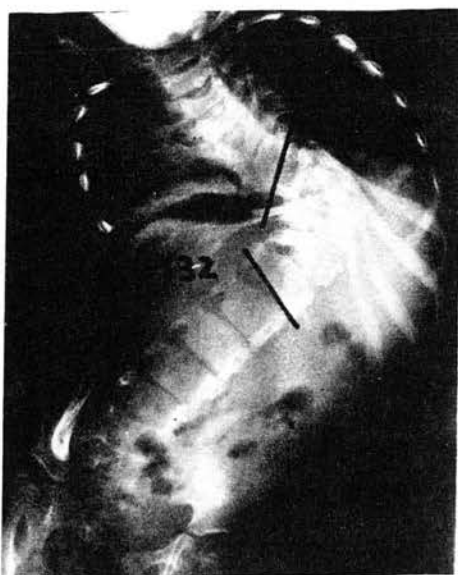


Fig. 3A. Antero-posterior radiograph of a 132 degree deformity associated with a unilateral bar.

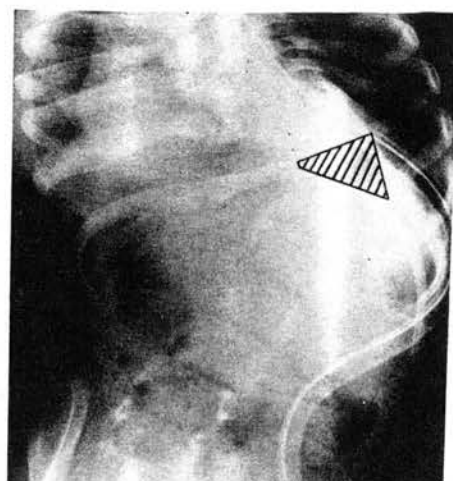


Fig. 3B. Antero-posterior radiograph after removal of the apical vertebral body.

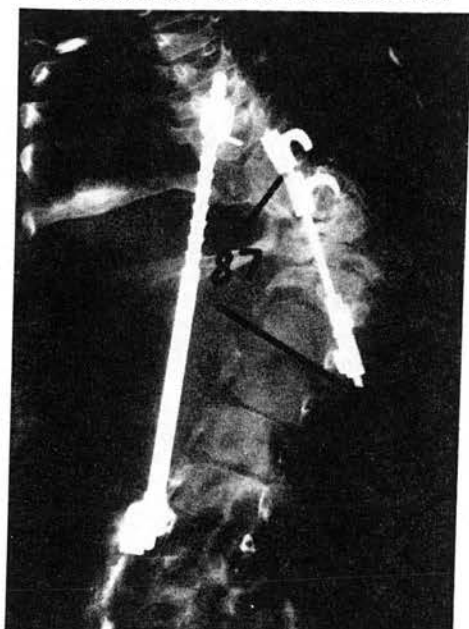


Fig. 3C. Antero-posterior radiograph after the second-stage posterior fusion to the sacrum with Harrington instrumentation.

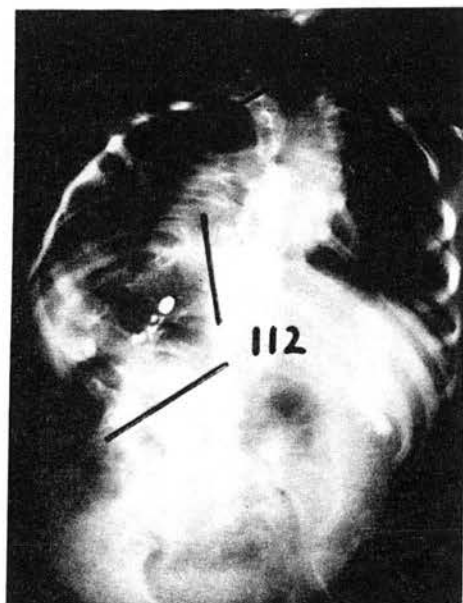


Fig. 4A. Antero-posterior radiograph showing severe double structural curves with a unilateral bar and marked pelvic tilt.

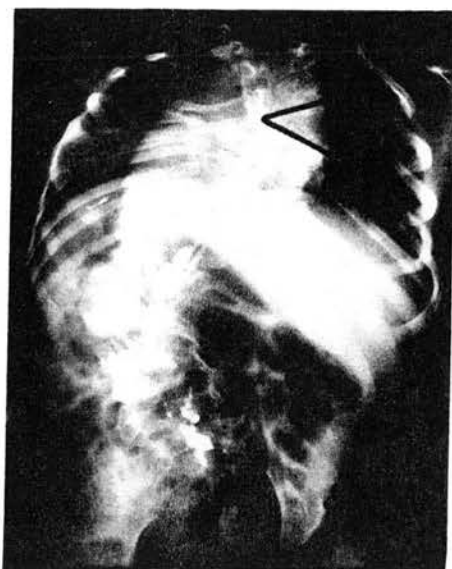


Fig. 4B. Antero-posterior radiograph after excision of the lower apical vertebral body.

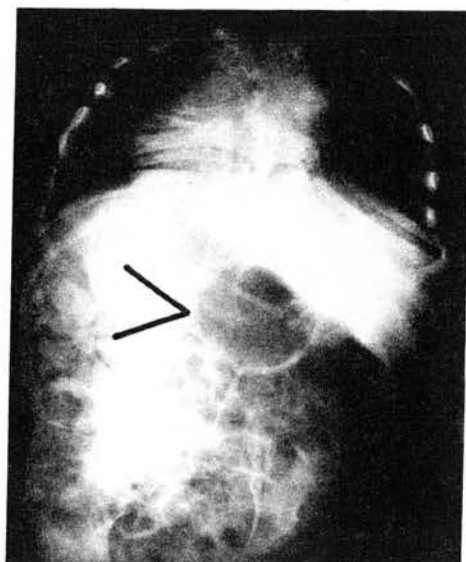


Fig. 4C. Antero-posterior radiograph after excision of the lower apical vertebral body.

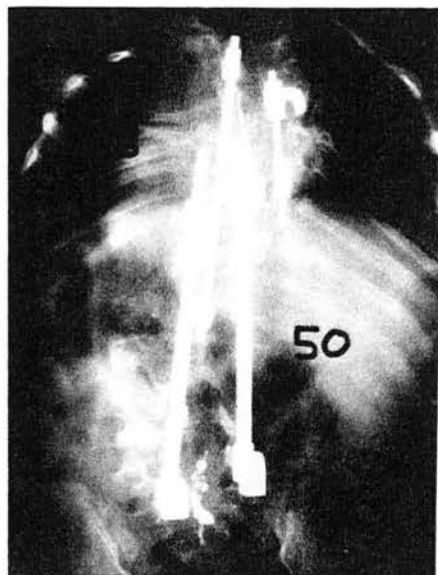


Fig. 4D. Antero-posterior radiograph after the third-stage posterior fusion with rods. The curves are markedly reduced, the chest is much more symmetrical, and the pelvic tilt is almost completely corrected.

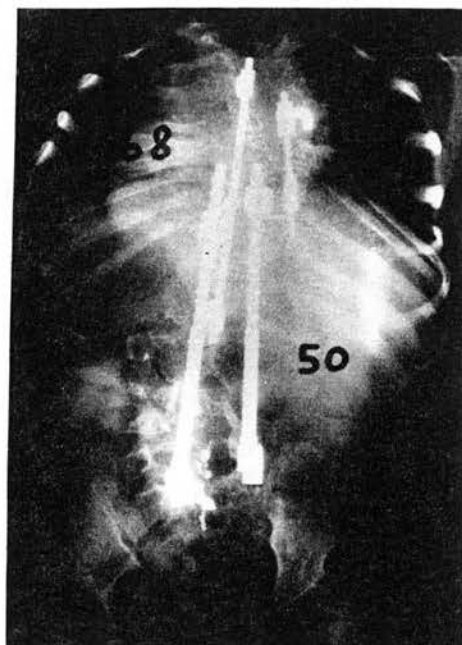


Fig. 4E. Antero-posterior radiograph at follow-up, one year later. Hooks and rods are holding and there has been no loss of correction.

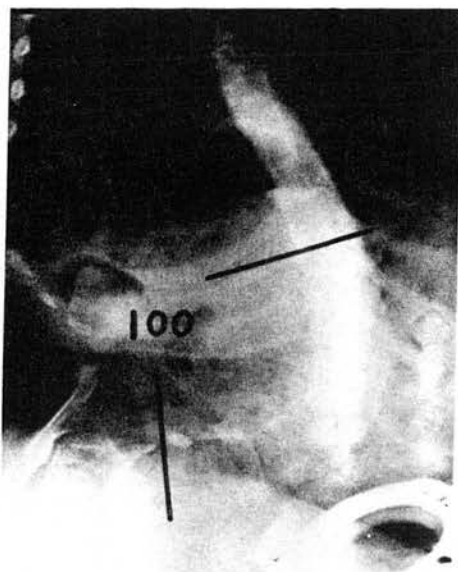


Fig. 5A. Antero-posterior radiograph showing a severe 100 degree paralytic curvature with marked pelvic tilt.

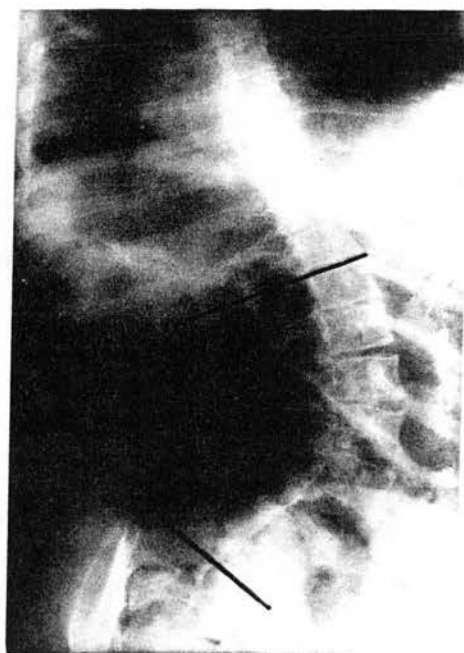


Fig. 5B. Antero-posterior radiograph after halo-femoral traction. The curvature has been reduced to 60 degrees.

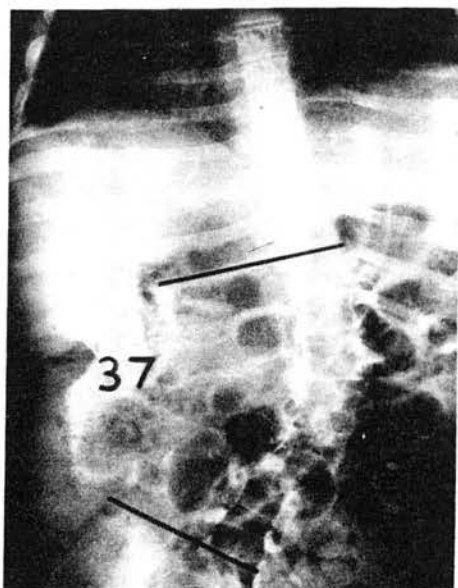


Fig. 5C. Antero-posterior radiograph after excision of the third lumbar vertebral body. The curvature has been reduced further to 37 degrees and a spontaneous interbody fusion has occurred.

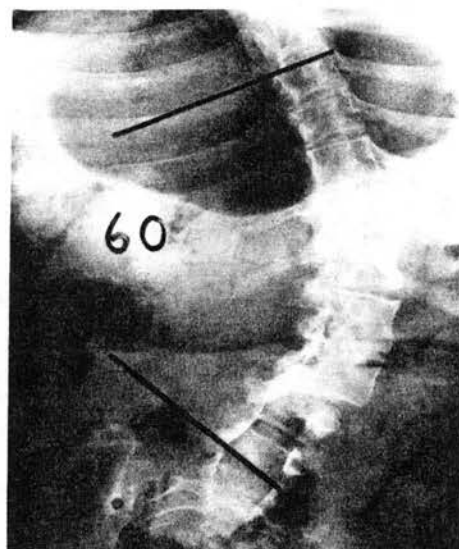


Fig. 5D. Antero-posterior radiograph two years later. The deformity has regressed to 60 degrees.

CONGENITAL LUMBAR KYPHOSIS IN MYELOMENINGOCELE - VERTEBRAL BODY
RESECTION AND POSTERIOR FUSION.

Leatherman KD, Dickson RA.

Spine; 3: 222-226, 1978.

Congenital Kyphosis in Myelomeningocele

Vertebral Body Resection and Posterior Spine Fusion

KENTON D. LEATHERMAN, MD, FACS,*
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The deforming mechanisms in the congenital lumbar kyphosis of myelomeningocele are situated anteriorly. Posterior wedge resection with local fusion therefore will not prevent progression of the deformity. Nonetheless, this procedure may be the only method of affording sac closure, or providing continuity of the ulcerated integument. Definitive corrective surgery will be required later and must combat the anterior deforming forces. Resection of the apical vertebral body followed by posterior fusion with Harrington instrumentation provides a solid straight spine. [Key words: myelomeningocele, congenital kyphosis, vertebral body resection, spine fusion, Harrington rod]

WHILE PARALYTIC spinal deformities are frequently seen in patients with myelomeningocele, congenital kyphosis is less common, accounting for less than 10% of spinal deformities in these individuals.⁵ Conservative management is of no avail in these severe, rigid, and progressive lumbar deformities which are present at birth. Surgical intervention is, therefore, frequently necessary and is indicated to provide sitting stability in these individuals, the majority of whom are destined to be nonwalkers. Sharrard has developed a technique of neonatal osteotomy-excision which is performed on the first day of life and is a most useful way of providing skin cover of good quality without tension.³ Unfortunately, many spinal centers do not see these children until much later in life when established skin ulceration and progression of deformity are the rule rather than the

exception. Posterior wedge resection has again been the recommended management in this situation.^{4,5} However, after initial gratifying correction of the deformity, many continue to progress. This is because the deforming forces are situated anteriorly while the surgical attack has been purely posterior. We have developed a technique of shortening the spine as well as straightening it for the correction of severe rigid deformities,² and have applied it to the problem of the congenital kyphosis in myelomeningocele.

PATHOLOGIC ANATOMY

A clear understanding of the deforming mechanisms in the congenital lumbar kyphosis of myelomeningocele is essential. These comprise both bony and soft tissue abnormalities. Figure 1A is a lateral radiograph of the spine of a 4-month-old female and shows the characteristic kyphotic deformity. The apical vertebral body is wedged anteriorly and also, to a lesser extent, the vertebra above. These patients also have deficient posterior elements, and these deficits may be extensive with a high level lesion. Therefore, anterior structural change and

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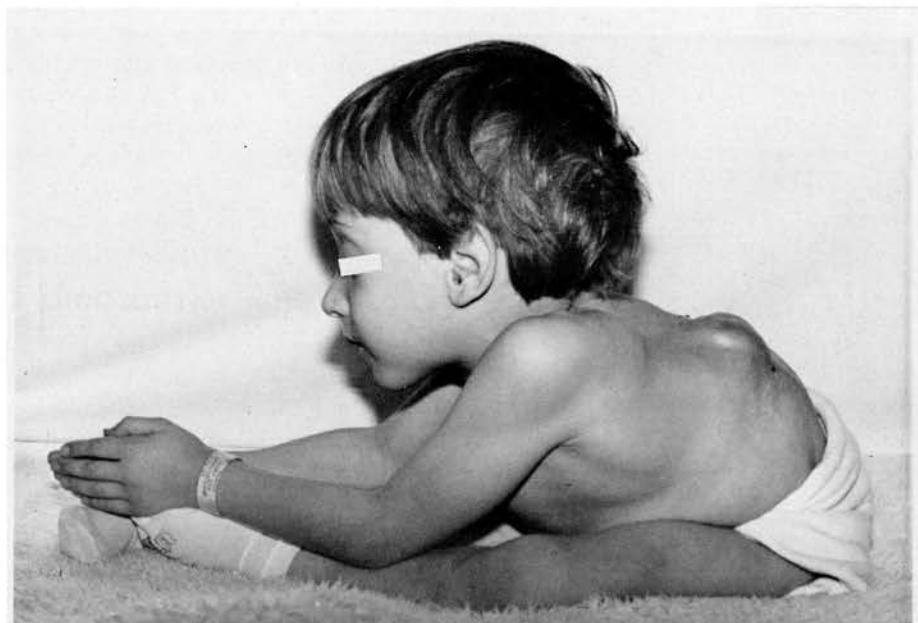
Fig 1A. Lateral radiograph of the spine of a 4-month-old infant with the congenital lumbar kyphosis of myelomeningocele. **B.** Lateral radiograph of same patient 3 years later, showing marked progression of the deformity.



posterior structural insufficiency are present coincidentally. A further bony abnormality exists in relation to this deformity and is largely responsible for the associated soft tissue changes which adversely affect the spines of these patients. The abnormal laminae and deficient spinous processes are so splayed that the posterior surfaces face anteriorly. The posterior paravertebral musculature which, under normal circumstances, would exert an extensor influence on the spine, now comes to lie anteriorly with a subsequent flexor effect, further aggra-

vating the existing structural deformity. The psoas muscle and the crura of the diaphragm are additional offenders. Dissected specimens have demonstrated that a tight anterior longitudinal ligament and annuli fibrosi are contributory factors.¹ Figure 1B, a lateral radiograph of the same child taken 3 years later, illustrates this point. There has been marked progression of the deformity with the thoracic spine tilted forward and the sacrum almost horizontal. This has occurred without the further adverse influence of the other great deform-

Fig 2. Seven-year-old child with congenital lumbar kyphosis.



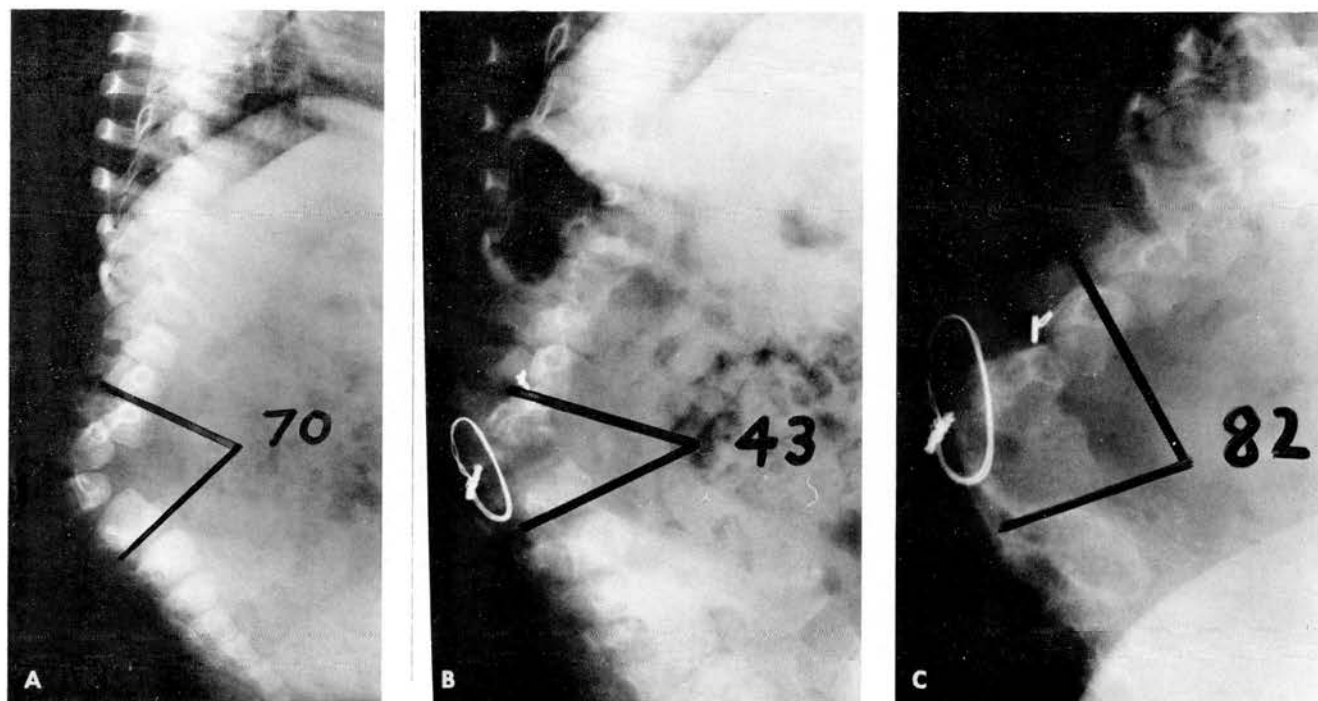


Fig 3A. Preoperative lateral radiograph of a patient with congenital lumbar kyphosis of 70°. **B.** Postoperative lateral radiograph of same patient showing a good initial correction. **C.** Lateral radiograph taken 10 months after surgery of same patient showing marked progression of the deformity.

ing force, gravity, since both these radiographs were taken in the supine position. The functional consequence for the individual is that the entire spine above the kyphosis angulates further forward until chest and thighs are approximated or the child topples over if he can sit at all (Figure 2).

Osteotomy-excision with limited fusion and internal fixation is destined to failure because the anterior deforming forces are not overcome. Figure 3 is an example of such a situation. A congenital lumbar kyphosis of 70° was treated by osteotomy-excision and posterior fusion with cerclage wires (Figure 3A). Initial correction of the deformity was gratifying (Figure 3B), but within a year, despite a sound fusion locally, the anterior deforming forces drew the spine into even more kyphosis than was present initially (Figure 3C). The concept of vertebral body excision and posterior fusion of the entire structural curve with Harrington instrumentation was therefore applied to this deformity to overcome the problem of the anterior deforming forces.

CASE REPORTS

Case 1. PF, aged 11 years and 3 months, with a congenital lumbar kyphosis measuring 90°, had a high neurologic level of paraplegia, well above the apex of the deformity. Due to the deformity this child, who was a nonwalker, had no sitting stability. Vertebral body resection and posterior fusion with Harrington instrumentation was performed as a one-stage procedure through a posterior approach. The redundant functionless spinal

cord was excised to expedite the procedure and enhance the fusion. The apical vertebral body was excised and sufficient bone above and below resected to eliminate tension on the anterior deforming forces and allow complete correction of the kyphosis. The patient's spine was fused with the aid of Harrington instrumentation well above and below the area of kyphosis. By this technique, an excellent correction of the deformity was obtained, and at follow-up 1 year later, hooks and rods were holding, a solid fusion was observed, and the child was an independent sitter. No complications were observed. This sequence of events is summarized in Figure 4.

Case 2. MS, aged 10 years and 2 months, had a neurologic level of involvement just below the apex of the deformity which measured 100° (Figure 5A). The spinal cord was thus not functionless at this level and could not be resected. The procedure was therefore performed in two stages. In the first stage, the apical vertebral body was excised by an anterior retroperitoneal approach. The anterior longitudinal ligament and annuli fibrosi were divided and excised at multiple levels. The apical vertebral body was then resected, and it was noted that the spinal deformity was now passively correctable, the anterior deforming forces having been overcome. The deficit after vertebral body resection was bridged by anterior strut grafting. The graft was secured by slotting it into the vertebrae above and below, and the patient was nursed with due respect for potential spinal instability. In the second stage performed 3 weeks later, the spine was fused posteriorly from the middle of the thoracic spine to the sacrum using Harrington instrumentation. The deformity was thereby corrected to 20° (Figure 5B), and at follow-up 4 years later, the

Fig 4A. Lateral radiograph of the spine of patient in Case 1 before corrective surgery. **B.** Lateral radiograph of same patient 1 year after surgery, showing a solid fusion and a good correction of the deformity.

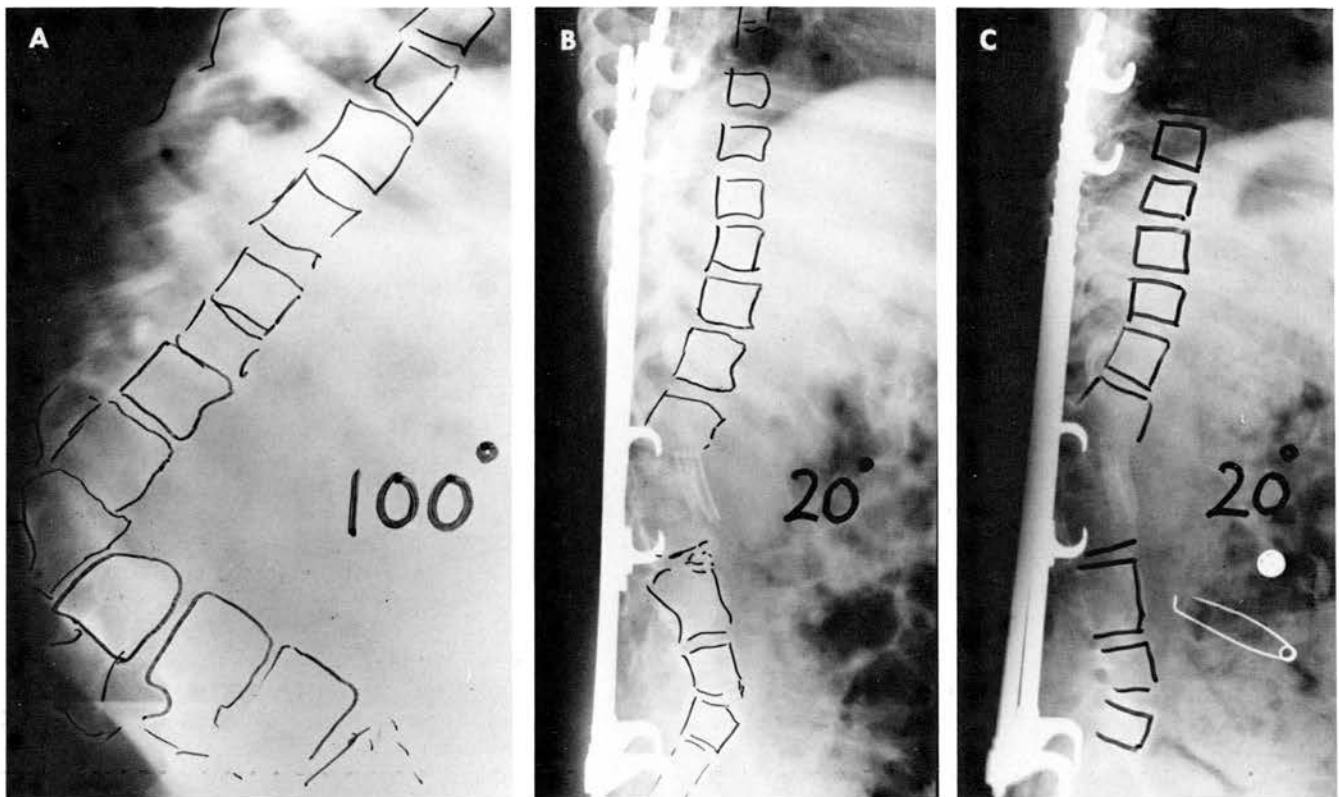
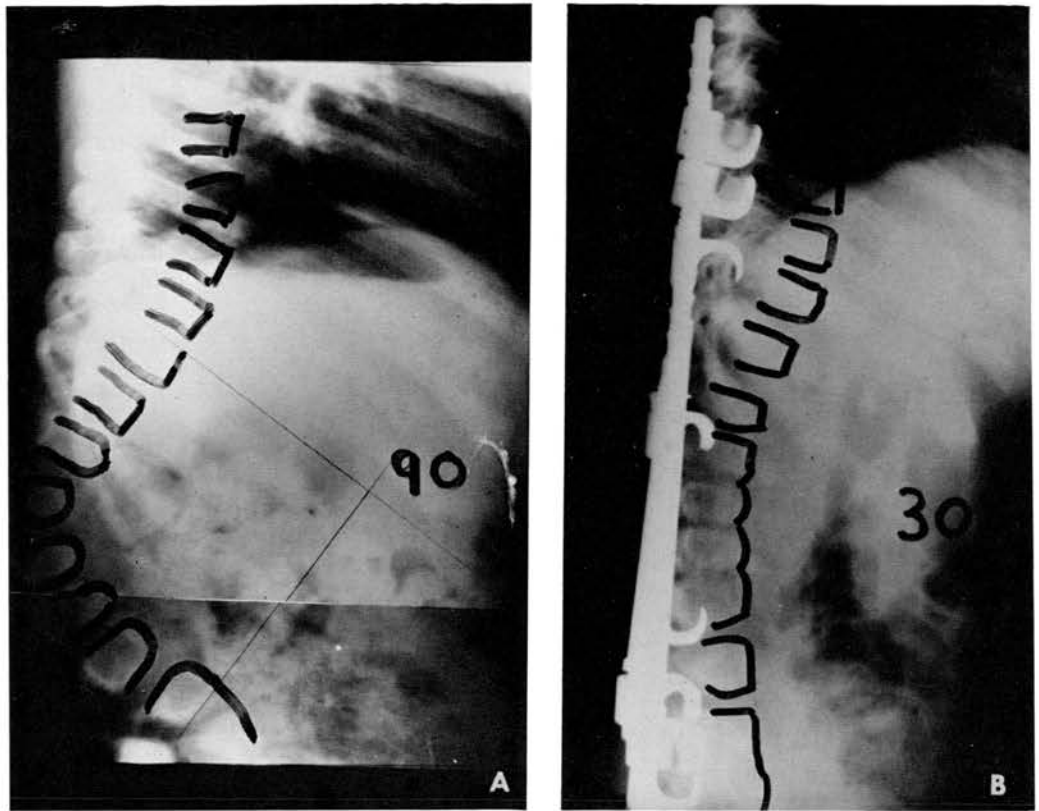


Fig 5A. Lateral radiograph of spine of patient in Case 2 showing kyphotic deformity of 100° before corrective surgery. **B.** Same patient after surgery, with correction of deformity to 20°. **C.** Same patient 4 years after surgery, showing maintenance of correction.

correction was sustained. The anterior graft had incorporated, and there was a solid posterior fusion (Figure 5C). No complications occurred with this procedure, and the child is an independent sitter with a clinically straight spine.

DISCUSSION

Congenital lumbar kyphosis in myelomeningocele is characterized by its severity, rigidity, and progression. Superimposed on the structural vertebral changes are strong anterior soft tissue deforming forces. The sitting potential of these children, many of whom are already destined to be nonwalkers, is in serious jeopardy. Furthermore, problems concerning the quality of the skin overlying the kyphosis are an ever-present threat to the neurologic level and life itself, there being a high incidence of cerebrospinal infection in these individuals.⁴

Osteotomy-resection may solve the immediate problem of providing a good quality to the overlying integument and this should be the initial treatment of choice. Such a procedure, however, cannot be expected to prevent progression of deformity, as the anterior deforming forces have not been overcome. Posterior vertebral body resection and posterior spine fusion with Harrington instrumentation have been shown to be successful in the treatment of congenital lumbar kyphosis in myelomeningocele.⁵ It is important to emphasize that this procedure can only be performed satisfactorily as a one-stage posterior operation when the spinal cord can be sacrificed with certainty. A two-stage anterior and then posterior approach has already proved itself effective in the treatment of other severe, rigid, spinal deformities,² and is the only method of dealing directly with the anterior deforming forces, particularly when the cord cannot be sacrificed. Furthermore, the Harrington

instrumentation can be inserted with greater ease and with less tension on hook sites in already porotic bone when the spine has previously been made supple by the first stage procedure. This is supported by the presence of a solid fusion, with hooks and rods holding, and no loss of correction 4 years after surgery in one case reported herein. The procedures described are probably most suitable for children between ages 5 and 12.

REFERENCES

1. Hoppenfeld S: Congenital kyphosis in myelomeningocele. *J Bone Joint Surg* 49B:276-280, 1967
2. Leatherman KD: The management of rigid spinal curves. *Clin Orthop* 93:215-224, 1973
3. Sharrard WJW: Spinal osteotomy for congenital kyphosis in myelomeningocele. *J Bone Joint Surg* 50B:466-471, 1968
4. Sharrard WJW, Drennan JC: Osteotomy-excision of the spine for lumbar kyphosis in older children with myelomeningocele. *J Bone Joint Surg* 54B:50-60, 1972
5. Sriram K, Babechko WP, Hall JE: Surgical management of spinal deformities in spina bifida. *J Bone Joint Surg* 54B:666-676, 1972

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COTREL TRACTION, EXERCISES, CASTING IN THE TREATMENT OF IDIOPATHIC
SCOLIOSIS - A PROSPECTIVE CONTROLLED CLINICAL TRIAL.

Dickson RA, Leatherman KD.

Acta Orthopaedica Scandinavica; 49: 46-48, 1978.

COTREL TRACTION, EXERCISES, CASTING IN THE TREATMENT OF IDIOPATHIC SCOLIOSIS

A Pilot Study and Prospective Randomized Controlled Clinical Trial

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A pilot study of ten individuals with adolescent-onset idiopathic scoliosis demonstrated that a week of Cotrel traction and exercises did not improve curve correction obtained by the application of an elongation, derotation, flexion (EDF) cast. There was, however, a significant improvement in lateral bending correction during this period. A prospective, randomized, controlled clinical trial showed that the exercise programme and not the traction was responsible for rendering the spine less rigid.

Key words: scoliosis; traction; exercises; casts; lateral bending

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The scoliosis treatment team in this centre was impressed by the management of scoliosis as demonstrated by Cotrel & Morel (1964) when they visited Berck-Plage. Subsequently Cotrel visited Louisville and demonstrated his technique of traction, exercises, and casting (elongation, derotation, flexion-EDF). Since then we have used this method for the non-operative treatment of idiopathic scoliosis but have been disappointed with the long-term results. Furthermore it is time-consuming in terms of manpower and hospitalization.

Nachemson & Nordwall (1976, 1977) demonstrated that Cotrel traction is of no value in the preoperative phase of treatment of idiopathic scoliosis. Their patients did not achieve a better operative correction of their curve nor did any benefit in terms of safety accrue when surgery was preceded by a week of traction. Ramsay et al. (1976) have shown

that with Cotrel traction curve correction can be obtained but that no further improvement is achieved after 8 days. Physical therapy is an integral part of this treatment programme and it is not clear whether the traction or "loosening-up" exercises, or indeed both, are responsible for curvature correction. A pilot study and a prospective randomized controlled clinical trial were therefore carried out.

PILOT STUDY

Patients and method

Ten consecutive patients with adolescent-onset idiopathic thoracic scoliosis were investigated. There were nine females and one male. Their mean age was 12.9 years. They underwent our usual in-patient treatment programme of traction and physical therapy exercises and were EDF casted on the eighth day after admission. Traction was in the

form of autoelongation and these individuals were encouraged to perform this throughout the day. At night the traction was altered to fixed traction starting with 5 lb rising to 12 lb on their seventh night. Physical therapy exercises were performed in the gymnasium for two 1-h periods each day under the supervision of a physical therapist. In each session 20 exercises were performed 15 times each in succession and the various exercise routines concentrated on lateral bending correction of the curve and pelvic tilting to mobilize the concomitant lumbar lordosis. On the eighth day an EDF cast was applied under maximum tolerable fixed traction. In order to control the investigation these individuals were similarly EDF casted on day 1 before treatment was commenced. This cast was then removed in order that they could go through the treatment regime.

A series of antero-posterior (AP) radiographs of the spine were taken during the treatment period and the curves were measured by the Cobb method (Cobb 1948). The variables studied were:

1. Standing curve before treatment
2. Curve on lateral bending before treatment
3. Standing curve in first cast, before treatment.
4. Curve on lateral bending before second cast.
5. Curve under maximum traction before second cast.
6. Standing curve in second cast, after treatment.

Results

The mean magnitude of deformity during standing before treatment was 43°; on lateral bending before treatment 20°; and standing curve in first cast 26°. Table 1 shows the effect of traction and exercises on the standing curve in the cast, and on lateral bending. There was no significant difference between the curve measurements in the casts on day 1 and day 8, 26° and 26° ($P > 0.05$). Lateral bending corrected the curve from 20° to 13°, a statistically significant correction at the 0.1 per cent level. In no case did the maximum traction correction equal or better the correction determined by initial lateral

bending, and cast correction was significantly worse than the maximum traction correction ($P < 0.05$).

Comment

From this pilot study it was considered that 8 days of hospitalization for traction and physical therapy was not justified if a policy of EDF casting was to be pursued, as no benefit in terms of curve correction in the cast occurred from such a treatment regime. However, it appeared that treatment had improved the lateral bending correction significantly and considerably. As the maximum traction correction after treatment did not even approach the initial lateral bending correction, physical therapy exercises seemed to be the most likely cause of the improvement in lateral bending correction. A prospective, randomized, controlled clinical trial was therefore carried out.

PROSPECTIVE, RANDOMIZED, CONTROLLED CLINICAL TRIAL

Patients and method

The next 20 patients with a diagnosis of adolescent onset idiopathic scoliosis were randomly allocated to one of two treatment groups:

1. Traction and no exercises
2. Exercises and no traction.

Traction or exercises were performed as in the pilot study.

The mean age of the two groups of patients did not differ significantly, 13.1 years and 13.6 years ($P > 0.05$) nor did mean curve magnitude before treatment, 42° and 40° ($P > 0.05$). The same variables were studied in these groups as in the original pilot study.

Results

1. *Traction only.* Table 2 shows the effect of traction on the standing curve in the cast and the curve on lateral bending. There was no significant difference between the cast measurements on day 1 and day 8, 32° and 35°, respectively ($P > 0.05$). Nor was there any

Table 1. Results of pilot study

	1	2	P
Standing curve in cast	26°	26°	> 0.05
Curve on lateral bending	20°	13°	< 0.001

Table 2. Results of trial with traction only

	1	2	P
Standing curve in cast	32°	35°	> 0.05
Curve on lateral bending	21°	22°	> 0.05

significant difference between the lateral bending corrections, 21° and 22°, respectively ($P > 0.05$). The mean correction in maximum traction, 27°, was significantly worse than the correction obtained by initial lateral bending ($P < 0.05$).

2. *Exercises only.* Table 3 shows the effect of exercises alone on the standing curve in the cast and the curve on lateral bending. There was again no significant difference between

Table 3. Results of trial with exercises only

	1	2	P
Standing curve in cast	32°	33°	> 0.05
Curve on lateral bending	20°	16°	< 0.001

the cast corrections on day 1 and day 8, 32° and 33°, respectively ($P > 0.05$). However there was a significant improvement in lateral bending, 20° and 16°, respectively ($P < 0.001$).

DISCUSSION

There appears little justification for a period of traction and physical therapy if the patient is to be EDF casted as there is no improvement in cast curve magnitude in this time. Furthermore the improvement in lateral bending so obtained is completely nullified by

holding the spine in a cast in a position far worse than even the degree of correction obtained by lateral bending before treatment started. In addition it would seem illogical, after making a spine less rigid, to imprison it in a cast. However, the trial clearly demonstrates that traction and exercises confer no benefit over exercises alone and that physical therapy is the likely cause of the improved lateral bending. Perhaps the most sensible therapy should be a period of intensive physical therapy followed by a Milwaukee brace about which considerably more is known and with which the majority of spinal surgeons are at least familiar. Coming out of the brace for short periods to perform an exercise programme would appear justified and thus the spine can be stretched to its limits which clearly cannot occur in a cast.

REFERENCES

- Cobb, J. R. (1948) *Outline for the study of scoliosis*. Instructional Course Lectures, the American Academy of Orthopaedic Surgeons, Vol. 5, p. 261. J. W. Edwards, Ann Arbor.
- Cotrel, Y. & Morel, G. (1964) La technique de L'E.D.F. dans la correction des scolioses. *Rev. Chir. orthop.* 50, 59–75.
- Nachemson, A. & Nordwall, A. (1976) The Cotrel dynamic spine traction—an ineffective method for preoperative correction of scoliosis. *J. Bone Jt Surg.* 58-A, 158.
- Nachemson, A. & Nordwall, A. (1977) Effectiveness of preoperative Cotrel traction for correction of idiopathic scoliosis. *J. Bone Jt Surg.* 59-A, 504–508.
- Ramsey, P. L., Wickersham, J., Kingsbury, H. & Lou, D. (1976) Mechanical analysis of Cotrel traction in idiopathic scoliosis. *J. Bone Jt Surg.* 58-A, 157.

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TWO-STAGE CORRECTIVE SURGERY FOR CONGENITAL DEFORMITIES
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TWO-STAGE CORRECTIVE SURGERY FOR CONGENITAL DEFORMITIES OF THE SPINE

KENTON D. LEATHERMAN, ROBERT A. DICKSON

From Kosair Scoliosis and Spine Treatment Center, Louisville

Sixty patients with congenital deformities of the spine were operated upon in the past fifteen years using a two-stage procedure. In the fifty patients with scoliosis half of the deformities were due to hemivertebrae and half to unilateral bars. The average correction of the deformity was 47 per cent. Early neurological signs observed in two patients with a diastematomyelia resolved. Of the ten patients with kyphosis nine had neurological signs of impending paraplegia and one was completely paraplegic before operation; all improved markedly.

Posterior spinal fusion alone in the rapidly progressing congenital deformity may not prevent further progression, particularly in those cases with unilateral bars. Anterior resection of the vertebral body with later posterior fusion with Harrington instrumentation is safe and effective.

The Scoliosis Research Society has accepted the classification of spinal deformities proposed by Goldstein and Waugh (1973) which includes a classification of congenital deformities devised by MacEwan, Conway and Miller (1968) with subdivision into scoliosis, kyphosis or lordosis. The embryological aetiology may be a failure of bone formation, a failure of bone segmentation, or both. Failures of formation may be complete and unilateral (hemivertebrae) or partial and unilateral (wedge vertebrae). Failures of segmentation may be unilateral, producing "bars", or bilateral, producing "blocks". Long-term studies of the behaviour of these deformities emphasise the need for a clear understanding of their aetiology (Winter, Moe and Eilers 1968; Winter, Moe and Wang 1973; James 1975). Solitary hemivertebrae, solitary wedge vertebrae, or balanced hemivertebrae on each side of the spine are not usually associated with rapid progression. This is the case in approximately 50 per cent of congenital deformities of the spine. Multiple hemivertebrae on the same side, and particularly unilateral unsegmented bars, are, however, notorious for their progression, there being normal or near-normal growth on one side of the spine and no growth or virtually no growth on the other (Bradford, Moe and Winter 1975).

In the management of congenital deformities of the spine this asymmetry of growth potential must not be allowed to cause progression of the deformity: if progression is noted, treatment is indicated. Because external support by casts or braces is successful in only a small percentage of cases, an operation is the definitive treatment (Winter 1973). Harrington instrumentation is contraindicated due to the high incidence of paraplegia as a result of tethered spinal cords (Winter 1973). As a

consequence posterior "fusion-in-situ" has become the recommended procedure, even in very young individuals (Winter 1973), but it is at best only a holding operation. Furthermore, in rapidly progressive deformities, not only does progression continue, but it may do so at a greater rate due to the tethering effects of the fusion (Roaf 1966). This is hardly surprising as the fusion is posterior, some distance from the area of asymmetric growth which is anterior. In addition, these deformities are rigid.

In order to correct a rigid spine without incurring the danger of traction paraplegia, the spine must be shortened as well as straightened. A two-stage corrective procedure has been introduced for this purpose (Leatherman 1969, 1973). This paper describes the operative considerations and the results of sixty patients treated thus in this centre over the past fifteen years.

DEVELOPMENT OF THE TWO-STAGE PROCEDURE

The first report of the removal of a portion of the spine for a fixed deformity was by Royle (1928). A two-stage procedure was reported by von Lackum and Smith (1933): in the first stage the vertebral body and posterior elements were removed and in the second stage the spine was fused. Serious complications arose from such operations where the vertebral body and posterior elements were removed at the same time (Wiles 1951). A two-stage procedure was, therefore, conceived with the vertebral body being removed through an anterior approach in the first stage and the posterior elements removed and fusion performed in the second stage. With his interest in the anterior approach

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to the spine for tuberculosis, Hodgson (1965) reported two cases of fixed kyphosis corrected by anterior opening-wedge osteotomy and anterior strut grafts. He suggested that alternatively a closing-wedge osteotomy could be done on the convex side of the deformity. This forms the basis of the first stage of the two-stage procedure and enables the spine to be shortened as well as straightened to preserve the neurological function. As a result the anterior asymmetric growth is halted. However, the curve remaining above and below the osteotomy will continue to deform throughout growth. A posterior fusion of the entire curve is therefore necessary; this is the basis of the second stage, usually performed three weeks later when the anterior wound

pleura along the line of the rib to its attachment to the spine, thus allowing the entire costovertebral articulation to be seen. The pleura is then gently lifted upwards and downwards to expose the apical vertebral body and the bodies above and below. The segmental vessels at these three levels are then doubly ligated and divided. This should be performed on the anterior surface of the vertebral bodies so as not to impair the vertically orientated anastomosis which is important in the nutrition of the spinal cord. The periosteum is then incised in the same line as the pleura and these two layers afford a satisfactory closure at the completion of the resection. The rib attached to the apical vertebral body is now disarticulated at the costovertebral joint and its posterior 2 inches excised. The vertebra is now exposed ready for excision.

The thoracolumbar region of the spine is exposed by means of a thoraco-abdominal approach through the bed of the tenth or eleventh ribs. After rib resection the deep periosteum together with the pleura is incised throughout the length of the wound which is enlarged by

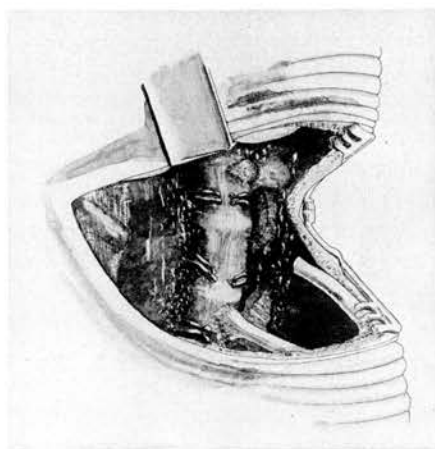


Fig. 1

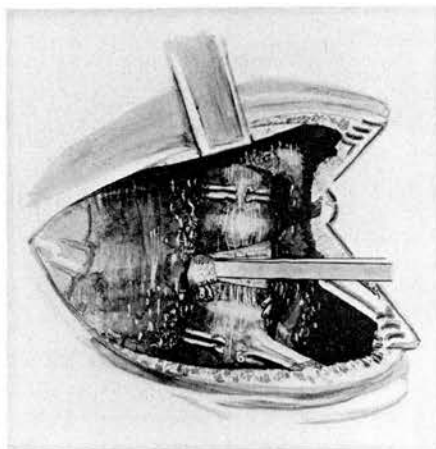


Fig. 2



Fig. 3

Operative technique. Figure 1—Exposing the vertebral column from the front through the chest. Figure 2—Resecting the vertebral body using sharp osteotomes. Figure 3—Completing the excision using pituitary punches.

has healed and the patient has recovered from the first operation. Between the two stages the patient is nursed free in bed without external support to the spine. In the second stage the posterior elements are removed at the same level to complete the wedge resection which is then closed with a compression system. Additional stability and correction are gained by the insertion of a distraction rod on the concave side.

OPERATIVE TECHNIQUE

Stage 1: anterior resection of the vertebral body. The apical vertebral body is excised and, depending on the location of the curve, thoracic, thoracolumbar, or lumbar exposures are necessary. These approaches have been well described (Riseborough 1973) but certain points of technique are most important when resecting a vertebral body. The spine is approached from the convex side of the curve which greatly facilitates exposure of the scoliotic spine which, in severe cases, may lie in contact with the rib cage or abdominal wall. Exposure of the kyphotic spine from the front is more exacting.

When approaching the thoracic spine exposure is facilitated by entering the chest through the bed of the rib above the vertebral body that is to be excised. This eliminates the overhang provided by the rib above. The spine is best located by dissecting the rib back and disarticulating it from its transverse process (Fig. 1). When there is adherence or rib fusion, disarticulation may be eased by incising the

means of a rib spreader. The diaphragm is detached peripherally leaving a fringe for reattachment at the end of the operation. The abdominal part of the exposure is entirely retroperitoneal, the peritoneum and its contents being mobilised by blunt dissection towards the concavity. An excellent view of the thoracolumbar region of the spine is thereby obtained and the periosteum and segmental vessels are dealt with as in the thoracic approach.

Exposure of the lumbar spine is achieved by the extraperitoneal approach used for lumbar sympathectomy. The peritoneum is displaced medially forwards off the posterior abdominal wall, the ureter and genital vessels being raised with it. The periosteum and segmental vessels are dealt with as before. The spine thus approached must be exposed laterally and anteriorly.

The apical vertebral body is resected by a closing-wedge osteotomy, more of the convexity being removed than of the concavity (Fig. 2). Thin layers of bone are removed with a sharp chisel or gouge. Bleeding from the richly vascular cancellous bone of the vertebral body is controlled by means of bone wax. The greater part of the vertebral body can thus be removed before the spinal canal is breached. Entry into the spinal canal is best gained by enlarging the intertransverse foramen after removal of the transverse process by rongeur. The posterior longitudinal ligament with a thin shell of cancellous bone attached is then removed by pituitary punches (Fig. 3). Bleeding is controlled by thrombin-soaked Gelfoam and cottonoid pledgets. The pedicle, transverse process, and facets on the concave side are left intact and provide stability to the spine between the two stages. The resection area is smoothed by rongeur, filled with Gelfoam soaked in thrombin and closed by suturing the pleura and periosteum.

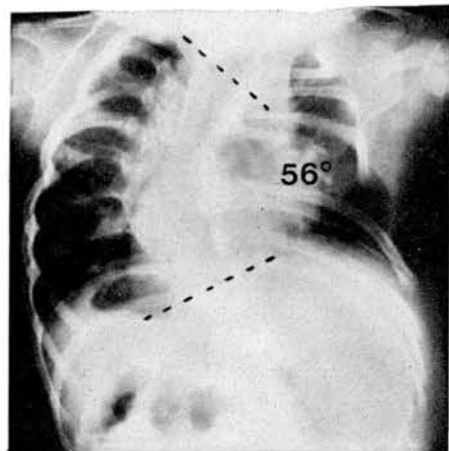


Fig. 4

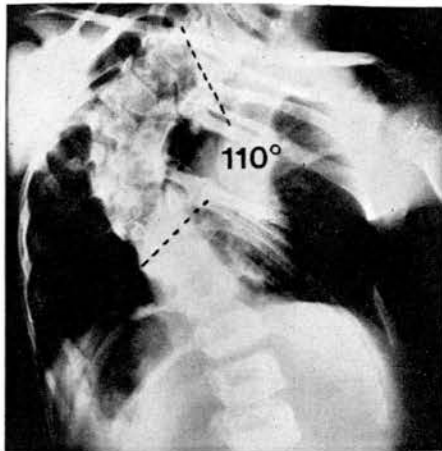


Fig. 5

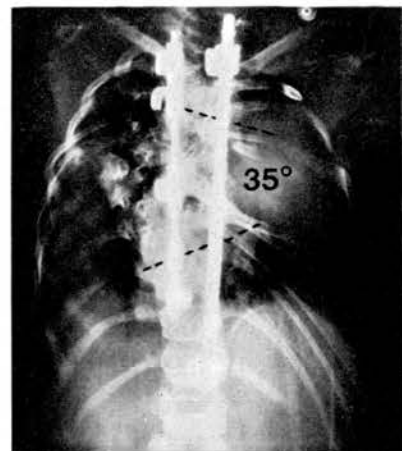


Fig. 6

Figure 4—A thoracic scoliosis of 56 degrees due to a unilateral unsegmented bar in a girl age one year two months. Figure 5—After posterior "fusion-in-situ" the deformity has progressed and now measures 110 degrees. Figure 6 After resection of the vertebral body and posterior fusion with Harrington instrumentation the deformity has been reduced to 35 degrees with restoration of chest symmetry and trunk balance.

Stage 2: posterior resection, fusion and instrumentation. The spine is approached posteriorly in the midline and the resection area identified. The posterior elements and the pedicle and transverse process on the concave side are removed to complete the closing wedge. This area is inspected to ensure that there are no sharp bony spicules or loose fragments. The wedge deficit is then closed by means of a compression system applied to the convex side and additional stability and correction obtained by inserting a distraction rod on the concave side. There must, however, be no strong distraction such as might embarrass the spinal cord or nerve roots. Posterior fusion of the entire structural curve is then performed. The patient is kept supine in a cast or a crutch type brace for six months; walking is then allowed but the spine is supported by this same well-fitting brace for a further six months.

CASE REPORT

Case 1. This girl, aged one year two months, had a congenital thoracic scoliosis of 56 degrees due to a unilateral bar (Fig. 4) and underwent a posterior "fusion-in-situ" of the entire curve. She was referred to this centre at the age of five years four months with a deformity of 110 degrees (Fig. 5). Posterior fusion had not halted progression and may have accelerated it. Studies of pulmonary function showed a severe restrictive defect but there were no neurological deficits and myelography revealed no abnormality. Two-stage correction was performed but in the second stage the previous fusion mass was osteotomised in five places to facilitate adequate correction. The deformity was thereby corrected to 35 degrees with restoration of chest symmetry and trunk balance (Fig. 6). The patient made an uneventful recovery.

CLINICAL MATERIAL

Sixty patients with congenital deformities of the spine have been treated in this manner with a minimum follow-up of eighteen months. There were fifty with scoliosis and ten with kyphosis.

Congenital scoliosis

Patients and methods. There were thirty girls and twenty boys, the mean age at operation being eleven years (range two years three months to sixteen years eight months) and the mean follow up five years five months (range one year six months to twelve years seven

months). Half of the deformities were due to hemivertebrae and half to unilateral bars. Pulmonary function studies were performed on all patients before operation and, although diminution in pulmonary function did not correlate with the magnitude of the curve, it was observed that in thoracic curves the pulmonary function was never normal while in lumbar curves it was always normal. Myelography was performed routinely and a diastematomyelia was found in four patients, two of whom had clinical evidence of early neurological deficit.

Table I. Results of the two-stage procedure

	Before	After	Follow-up
<i>Scolioses</i>			
Mean curve (degrees)	76.7	41.0	42.8
Standard deviation	74.5	15.4	15.5
<i>Kyphoses</i>			
Mean curve (degrees)	72.2	39.7	41.6
Standard deviation	20.1	13.7	14.1

These bony spurs were removed in a separate procedure. The deformity was measured on a standing radiograph before operation, on a supine radiograph after the second stage, and on a standing radiograph at the follow-up examination (Cobb 1948).

Results. The measurements of the curves are shown in Table I. The mean correction obtained after the second stage was 47 per cent. At follow-up the mean curve was 43 degrees, but the large standard deviations indicate the wide range of curves treated. The early neurological signs observed in the two patients with a diastematomyelia resolved, and the following case illustrates such a patient.

Case 2. This girl, aged eight years one month, had a congenital scoliosis measuring 95 degrees due to a unilateral bar (Fig. 7) and early neurological signs of impending paraplegia. A myelogram revealed a diastematomyelia at the apex of the curve (Fig. 8). After a two-stage corrective operation including removal of the bony spur, the deformity was reduced to 26 degrees (Fig. 9) and her neurological signs disappeared.

Congenital kyphosis

Patients and methods. There were four girls and six boys, the mean age at operation being twelve years three months (range two years seven months to twenty-nine years three months) and the mean follow-up three years eight months (range fourteen months to ten years). Eight of the deformities were due to hemivertebrae, one to a wedge vertebra, and one to an anterior bar, and all were at the thoracolumbar junction. Nine patients had neurological signs of impending paraplegia and one was completely paraplegic before operation. Myelography

COMPLICATIONS

Early complications comprised three cases of delayed healing, two of atelectasis, and one pleural effusion, making a total of six, or 10 per cent of all cases. These less severe problems resolved uneventfully. There was only one late complication, a pseudarthrosis, but there were no neurological problems, no wound infections, and no deaths in this series.

DISCUSSION

Posterior spinal fusion is successful in preventing progression of adolescent idiopathic curves because the potential for asymmetric growth is not great. This, however, is not so in rapidly progressive congenital deformities, particularly those due to unilateral unsegmented bars where the potential is at a maximum and a posterior "fusion-in-situ" may not prevent progression

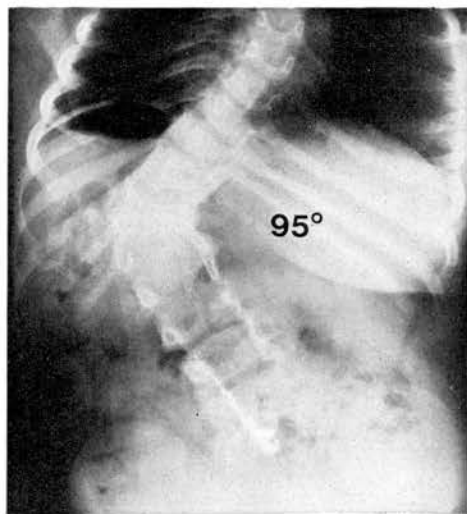


Fig. 7



Fig. 8

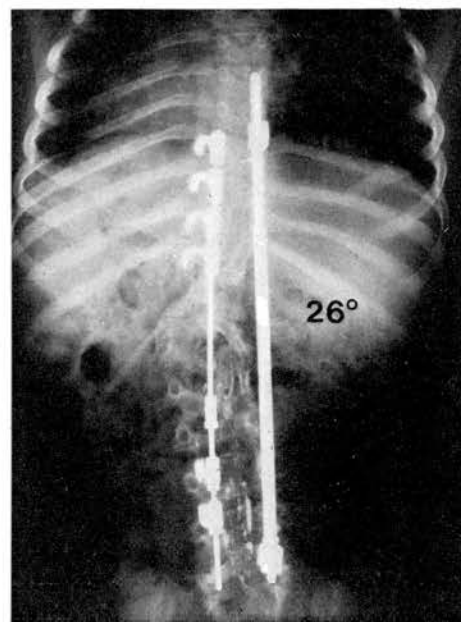


Fig. 9

Case 2
Figure 7—A congenital scoliosis of 95 degrees due to a unilateral bar in a girl aged eight years one month with neurological signs of cord compression. Figure 8—Myelogram showing a diastematomyelia. Figure 9— After the two stage procedure including removal of the diastematomyelia the deformity was reduced to 26 degrees. The neurological signs disappeared.

performed in all cases showed evidence of compression of the cord from the front. The curve was measured as for the scoliosis patients.

Results. The size of the curves and the corrections (Table I) were similar to those for the scoliosis group but were of less importance as these individuals were operated upon because of their neurological signs. The nine patients with early neurological deficit recovered completely, while the patient with paraplegia recovered sensation and control of the bladder but only minimal motor function. At follow-up he was able to walk with long leg braces and crutches. This case has already been described in detail (Leathermann 1973).

(Figs 4 and 5). Furthermore, as these deformities develop they also become rigid. Neurological problems are easily produced by attempting to correct them with Harrington instrumentation alone. The only way of correcting a rigid deformity without incurring the danger of neurological complications is to shorten the spine as well as to straighten it. The development of anterior resection as the initial part of a two-stage technique is particularly relevant when there are coexistent neurological problems, for these can only be solved by an anterior approach. Hall (1973) stated that a spinal cord compressed from the front by a kyphosis or any other cause must be decompressed from the front as a

laminectomy will not only be futile but actually harmful.

Patients with congenital deformities of the spine need a very thorough evaluation before operation. This should include a careful neurological examination and myelography is mandatory. When dealing with the thoracolumbar junction or the lumbar spine, intravenous pyelography is essential so that any associated congenital renal abnormality can be appreciated before operation. Pulmonary function studies are performed routinely and varying degrees of restrictive lung defects noted in thoracic curves. These spirometric tests are only a part of the evaluation and it is most important that these patients be examined by an expert in cardiopulmonary function. In cases where there is considerable

embarrassment of heart and lung function the assistance of such an expert during the postoperative phase is most helpful. Similarly, these patients should be carefully assessed by the anaesthetist. When the anterior surgical approach can be expected to be particularly exacting, as in a case of marked kyphosis at the thoracolumbar junction, we have no hesitation in obtaining the help of an experienced general surgeon when exposing the spine. Similarly, when a diastematomyelia is resected the assistance of a neurosurgeon should be sought by those unfamiliar with this procedure.

By such scrupulous attention to detail in the planning of the operative procedure and its performance, complications are kept to a minimum.

REFERENCES

- Bradford, D. S., Moe, J. H., and Winter, R. B. (1975) Scoliosis. In *The Spine*, Volume 1, pp. 271-385. Edited by R. H. Rothman and F. A. Simeone. Philadelphia, London, Toronto: W. B. Saunders Company.
- Cobb, J. R. (1948) Outline for the study of scoliosis. *American Academy of Orthopaedic Surgeons, Instructional Course Lectures*, **5**, 261-275.
- Goldstein, L. A., and Waugh, T. R. (1973) Classification and terminology of scoliosis. *Clinical Orthopaedics and Related Research*, **93**, 10-22.
- Hall, J. E. (1973) Editorial comment: Newest view scoliosis. *Clinical Orthopaedics and Related Research*, **93**, 2-3.
- Hodgson, A. R. (1965) Correction of fixed spinal curves. *Journal of Bone and Joint Surgery*, **47-A**, 1221-1227.
- James, J. I. P. (1975) The management of infants with scoliosis. *Journal of Bone and Joint Surgery*, **57-B**, 422-429.
- Leatherman, K. D. (1969) Resection of vertebral bodies. *Journal of Bone and Joint Surgery*, **51-A**, 206.
- Leatherman, K. D. (1973) The management of rigid spinal curves. *Clinical Orthopaedics and Related Research*, **93**, 215-224.
- MacEwan, G. D., Conway, J. J., and Miller, W. T. (1968) Congenital scoliosis with a unilateral bar. *Radiology*, **90**, 711-715.
- Riseborough, E. J. (1973) The anterior approach to the spine for the correction of deformities of the axial skeleton. *Clinical Orthopaedics and Related Research*, **93**, 207-214.
- Roaf, R. (1966) *Scoliosis*, pp. 107-108. Edinburgh and London: E. & S. Livingstone Ltd.
- Royle, N. D. (1928) The operative removal of an accessory vertebra. *Medical Journal of Australia*, **1**, 467.
- von Lackum, LeR. H., and Smith, A. DeF. (1933) Removal of vertebral bodies in the treatment of scoliosis. *Surgery, Gynecology and Obstetrics*, **57**, 250-256.
- Wiles, P. (1951) Resection of dorsal vertebrae in congenital scoliosis. *Journal of Bone and Joint Surgery*, **33-A**, 151-154, 170.
- Winter, R. B. (1973) Congenital scoliosis. *Clinical Orthopaedics and Related Research*, **93**, 75-94.
- Winter, R. B., Moe, J. H., and Eilers, V. E. (1968) Congenital scoliosis, a study of 234 patients treated and untreated. *Journal of Bone and Joint Surgery*, **50-A**, 1-47.
- Winter, R. B., Moe, J. H., and Wang, J. F. (1973) Congenital kyphosis. *Journal of Bone and Joint Surgery*, **55-A**, 223-256, 274.

MANAGEMENT OF SPINAL DEFORMITIES.

Leatherman KD, Dickson RA.

John Wright: Bristol, 1988.

This comprehensive text book of spinal deformities correlates the scientific and therapeutic principles as applied to spinal deformities of all types. It was written by the second author with the bulk of the illustrative material being supplied by the first author.